

Annex to:

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## **Annex J – Evidence tables for observational studies on metabolic diseases including pregnancy endpoints**

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## Continuous variables related to the risk of obesity/abdominal obesity

### Continuous variables related to the risk of overweight/obesity: body weight, BMI, fat mass and derived indices

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
<b>Exposure: Total sugars</b>							
<b>1</b>	<b>NGHS</b>  USA  Lee et al. (2015)  6 y  Unclear funding	<b>N</b> = 2,379  <b>Population sampled:</b> Non-Hispanic Caucasian and African American girls with racially concordant parents from 3 sites  <b>Excluded:</b> pregnancy, pairs of observations where visits were <0.8 or > 1.2 years apart, implausible or invalid nutritional intake; and missing nutrition information, change in BMI, change in WC or other covariates.  <b>n</b> = 2,021 (5,156 pairs of observations)  <b>n at visits 2-3</b> = 1,597 <b>n at visits 3-4</b> = 1,415 <b>n at visits 4-5</b> = 1,304 <b>n at visits 7-8</b> = 840 <b>Ethnicity:</b> 51.1% Caucasian and 48.9% Black <b>Sex:</b> females <b>Age:</b> 9-10 y	<b>BMIz-score</b>  <b>Height and weight</b> were measured by research staff twice in accordance with standard protocols. A third measurement was taken if the difference was > 0.5 cm or >0.3 kg. The closest two of the three measures were used to calculate BMI. The 2000 Centers for Disease Control and Prevention growth charts were used to determine age-adjusted and sex-adjusted <b>BMI z-scores</b> .	<b>Tsp/d (mean ± SD)</b>  Visit 2: 25.8 ± 12.9 Visit 3: 27.2 ± 13.0 Visit 4: 26.3 ± 12.5 Visit 7: 28.0 ± 12.6  1tsp = 4 g  <b>Method:</b> 3-d DR	1-y change in <b>total sugar</b> intake vs 1-y change in BMIz-score  <b>Data collection:</b> every year. Each observation refers to two consecutive years.	<b>Model 1:</b> race; initial age, BMI, and puberty stage, parents' income, parents' education, dieting status, initial and change in physical activity and baseline sugar intake  <b>Model 2:</b> model 1 + initial and change in grams of fibre, percentage of energy from fat and percentage of energy from other carbohydrates  <b>Model 3:</b> model 2 + initial and change in total energy intake	A significant <b>positive</b> association between change in total sugars intake and change in BMI-z-scores over 1 y (model 2) became <b>non-significant</b> (model 3) after adjusting for total energy.  <b>Per each 1 tsp/d (4 g/d) increase</b> <b>B coefficients (95% CI)</b> <u>Model 1:</u> 0.001 (0.000, 0.002) <u>Model 2:</u> 0.002 (0.001, 0.002) <u>Model 3:</u> 0.001 (0.000, 0.002)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
2	<b>SCES</b>  Australia  Gopinath et al. (2013)  5 y  Mixed funding	<b>N</b> = 2,353  <b>Population sampled:</b> schoolchildren from Sydney  <b>Excluded:</b> NR <b>Follow-up rate:</b> 51.6%  <b>n</b> = 856 <b>Females:</b> 421 <b>Males:</b> 435  <b>Ethnicity:</b> 61.1% Caucasian, 19.5% East Asian, 4% Middle Eastern  <b>Age:</b> 12 y	<b>BMI and %BF</b> <b>Height</b> was measured with shoes off using a freestanding SECA height rod. <b>Weight</b> in kilograms was measured using a standard portable weighing machine, after removing any heavy clothing.  <b>%BF</b> A leg-to-leg body composition analyzer was used to estimate % BF by bioelectrical impedance analysis	<b>Baseline, g/d †</b>  <b>Females, mean ± SD</b> 129.2 ± 55.1  <b>Males (range)</b> <b>T1:</b> ≤120.91 <b>T2:</b> 121.1 – 143.7 <b>T3:</b> ≥143.8  <b>n</b> <b>T1:</b> 141 <b>T2:</b> 142 <b>T3:</b> 152  <b>Method:</b> SFFQ	<b>Total sugars</b> at baseline vs changes in BMI and %BF over the 5-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> age, ethnicity, parental education, passive smoking, change in energy intake, change in height, screen time and PAL	Non-significant ( <b>negative</b> ) associations were observed between the intake of total sugars at baseline and changes in BMI or %BF during the 5-y follow-up after adjustment for confounders in females (analysis with the exposure at baseline as continuous variable). In males (analysis by tertiles of the exposure at baseline), changes in BMI ( <b>positive</b> association) increased (p for trend= 0.09) and %BF ( <b>negative</b> association) <b>significantly</b> decreased across tertiles of sugar intakes (p for trend = 0.02).  <i>Reasons for the different analyses applied by sex are not given in the publication.</i>
3	<b>KoCAS</b>  South Korea  Hur et al. (2015)  4 y  Public funding	<b>N</b> = 811  <b>Population sampled:</b> children from four schools from city of Gwacheon  <b>Excluded:</b> Missing data for age, BMI or sugar intake. Daily energy intake <500 kcal or >4000 kcal; current treatment for hypertension, dyslipidemia, diabetes a disease that impacts body weight; attempting weight loss at baseline.  <b>Follow-up rate:</b> 79.6% <b>n</b> = 605 <b>Sex:</b> 48.3% females <b>Ethnicity:</b> Asian	<b>BMIZ-score and %BF</b> <b>Body weight</b> was measured without shoes or clothes using a body composition analyzer. <b>Height:</b> NR Age- and gender-specific <b>BMI z-scores</b> were calculated using the 2007 Korean National Growth Charts.  A leg-to-leg body composition analyzer was used to estimate <b>% BF</b> by bioelectrical impedance analysis.	<b>g/d (median (IQR))</b> 34.5 (23.5, 47.2)  <b>E% (median (IQR))</b> 8.3 (6.1, 10.7)  <b>Method:</b> 3-d DR	<b>Total sugars</b> at baseline vs BMIZ scores and %BF at the end of the 4-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> crude  <b>Model 2:</b> total energy and household income at baseline. Sex and age only for %BF.	Non-significant ( <b>positive</b> ) associations were observed between the intake of total sugars at baseline and BMIZ or %BF at follow-up.  <b>Per each 1 log (g/d) increase β coefficients (SE)</b>  <b>BMIZ score</b> <b>Model 1:</b> 0.04 (0.07) <b>Model 2:</b> 0.08 (0.09)  <b>%BF</b> <b>Model 1:</b> 1.04 (0.69) <b>Model 2:</b> 0.43 (0.66)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
		Age: 9-10 years					
<b>Exposure: free sugars and/or added sugars</b>							
<b>1</b>	<b>NGHS</b>  USA  Lee et al. (2015)  6 y  Unclear funding	<b>Study population and exclusion criteria as for total sugars</b>	<b><u>BMIz-scores</u></b> <b>Ascertainment of outcome as for total sugars</b>	<b>tsp/d (mean <math>\pm</math> SD)</b> Baseline: 21.0 $\pm$ 11.8 Follow-up 1: 22.3 $\pm$ 12.0 Follow-up 2: 22.1 $\pm$ 11.5 Follow-up 3: 22.6 $\pm$ 11.7  <b>Serving size:</b> 1 tsp = 4g  <b>Method:</b> 3-d DR	1-y change in <b>added sugar</b> intake vs 1-y change in BMIz-scores  <b>Data collection:</b> every year. Each observation refers to two consecutive years.	<b>Model 1:</b> race, initial age, initial BMI, initial puberty stage, parents' income, parents' education, dieting status, initial and change in physical activity and baseline sugars  <b>Model 2:</b> model 1 + initial and change in grams of fibre, percentage of energy from fat and percentage of energy from other carbohydrates  <b>Model 3:</b> model 2 + initial and change in total energy intake	A significant <b>positive</b> association between change in added sugars intake and change in BMI-z-scores over 1 y (model 2) became <b>non-significant</b> (model 3) after adjusting for total energy.  <b>Per each 1 tsp/d (4 g/d) increase</b> <b>B coefficients (95% CI)</b> <u>Model 1:</u> 0.001 (0.000, 0.002) <u>Model 2:</u> 0.002 (0.001, 0.002) <u>Model 3:</u> 0.001 (0.000, 0.002)
<b>1</b>	<b>QUALITY</b>  USA  Wang et al. (2014)  2 y  Public funding	<b>N = 630</b>  <b>Population sampled:</b> General population from Quebec with at least one biological parent that had obesity and/or abdominal obesity  At risk of obesity (at least one parent with obesity or central obesity)  <b>Excluded:</b> Diabetes, following a very restricted diet (< 2510 kJ/d), regular medication use, and serious psychological ailments.	<b><u>BMI and BF (kg)</u></b>  <b>Height</b> was measured using a stadiometer and <b>weight</b> using an electronic scale according to standardized protocols. Age- and sex-specific <b>BMI</b> percentiles were computed using the CDC growth charts <sup>1</sup> .  Participants were subcategorized into 2 groups: overweight/obese (BMI $\geq$ 85 percentile) and normal weight (BMI < 85 percentile).	<b>g/d from liquids sources (mean <math>\pm</math> SD)</b> 11.4 $\pm$ 12.5  <b>g/d from solids sources (mean <math>\pm</math> SD)</b> 40.4 $\pm$ 22.2  <b>Method:</b> Three 24-h DR	<b>Added sugars from liquid and solid sources</b> at baseline vs changes in BMI and BF over the 2-y follow-up  <b>Data collection:</b> exposure at baseline, outcome at baseline and end of follow-up	<b>Model:</b> baseline BMI (or baseline BF for this outcome), age, sex, tanner stage, energy intake, fat mass index and physical activity.	Non-significant <b>negative</b> associations between the intake of added sugars from either liquid or solid sources and changes in BMI or BF over follow-up  <b>Per each 10 g/d increase</b> <b>BMI, <math>\beta</math> coefficients (95% CI), kg/m<sup>2</sup></b> <b>Liquid sources</b> -0.005 (-0.128, 0.117) <b>Solid sources</b> -0.014 (-0.098, 0.070)  <b>BF, <math>\beta</math> coefficients (95% CI), kg</b> <b>Liquid sources</b>

<sup>1</sup> CDC. CDC growth charts: United States; 2000 [cited 2011 Oct 10]. Available from: <http://www.cdc.gov/nccdphp/dnpa/growthcharts/resources/sas.htm>.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
		<b>Follow-up rate:</b> 97% <b>n</b> = 472 <b>Sex:</b> 44.5 % females <b>Ethnicity:</b> Caucasian <b>Age:</b> 8-10 y	<b>BF</b> (kg) was terminated with DXA.				-0.041 (-0.288, 0.205) <b>Solid sources</b> -0.039 (-0.207, 0.130)
2	<b>DONALD</b>  Germany  Herbst et al. (2011)  6 y  Public funding	<b>N</b> = >1200  <b>Population sampled:</b> General population from Dortmund  <b>Excluded:</b> birthweight of <2500 g, less than 2 antropometric measurements at both age 0.5 and 7 y, implausible and/or incomplete 3-d dietary records, missing information on potential confounders.  <b>n</b> = 216  <b>Sex:</b> 48.6% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 1 y	<b>BMIz-score and %BF</b>  Length (up to 2 years), height, and body weight were measured by trained nurses according to standard procedures <sup>2</sup> . Sex- and age-independent <b>BMI</b> SD scores (or BMIz scores) were calculated using the German national reference data <sup>3</sup> .  <b>%BF</b> was calculated using data from the 4 skinfolds <sup>4</sup> (McCarthy, 2006), measured on the right side of the body at the biceps and triceps and subscapular and suprailiac sites to the nearest 0.1 mm with a Holtain caliper	<b>%E (median (IQR))</b> † 4.3 (1.8-7.9)  <b>Method:</b> 3-d DR	<b>Free sugars</b> at 1 y and changes in intake from 1 y to 2 years vs BMI-SDS and %BF at 7 y (end of follow-up)  <b>Data collection:</b> at 0.5, 1, 1.5 and 2 years, and every year until 7 years of age	<b>Model 1:</b> baseline characteristics (gestational age, birth year, anthropometric characteristics, breastfeeding), sex and animal protein intake at 1 y (or change in animal protein intake from 1 to 2 y for changes in free sugars intake)  <b>Model 2:</b> model 1 + paternal education (+ maternal overweight for %BF)  * Models include only variables that modified the regression coefficients in the unadjusted models by >10% or had a significant independent effect on the outcome	<b>Negative</b> associations between the intake of free sugars at 1 year and BMI-SDS ( <b>significant</b> ) and %BF at 7 y.  Non-significant ( <b>positive</b> ) associations between changes in free sugars intake between 1 and 2 y and BMI-SDS and %BF at 7 y.  <b>Per each 1 %E increase at baseline</b> <b>β coefficients ± SD</b>  <b>BMI-SDS</b> <b>Model 1:</b> -0.087 ± 0.056; p=0.1 <b>Model 2:</b> -0.116 ± 0.057; p=0.04  <b>%BF</b> <b>Model 1:</b> -0.008 ± 0.015; p=0.6 <b>Model 2:</b> -0.014 ± 0.015; p=0.4  <b>Per each 1 %E increase from 1 to 2 y</b> <b>β coefficients ± SD</b>  <b>BMI-SDS</b> <b>Model 1:</b> 0.062 ± 0.043; p=0.1

<sup>2</sup> WHO. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. WHO Technical Report Series 854. Geneva: WHO; 1995.

<sup>3</sup> Kromeyer-Hauschild K, Wabitsch M, Kunze D, et al. (2001) Percentiles of body mass index in children and adolescents evaluated from different regional German studies (article in German). Monatsschrift Kinderheilkd 149, 807–818.

<sup>4</sup> Deurenberg P, Pieters JJ, Hautvast JG. The assessment of the body fat percentage by skinfold thickness measurements in childhood and young adolescence. Br J Nutr. 1990;63:293–303.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
							<p>Model 2: <math>0.074 \pm 0.043</math>; <math>p=0.09</math></p> <p><b>%BF</b>            Model 1: <math>0.003 \pm 0.012</math>; <math>p=0.8</math>            Model 2: <math>0.002 \pm 0.012</math>; <math>p=0.8</math></p>
2	<b>Mr and Ms OS</b>  China  Liu et al. (2018)*  4 y  Public funding	<p><b>N</b> = 4,000</p> <p><b>Population sampled:</b> General population</p> <p><b>Excluded:</b> Unable to walk independently or with bilateral hip replacements, diabetes at baseline.</p> <p><b>Follow-up rate:</b> 75%</p> <p><b>n</b> = 3,421            Females = 1,714            Males = 1,707</p> <p><b>Ethnicity:</b> Asian</p> <p><b>Age:</b> <math>\geq 65</math> y</p>	<p><b>Body weight, BMI, BF (kg) and %BF</b></p> <p>Body weight was measured to the nearest 0.1 kg, with subjects wearing a light gown, using a physician balance beam scale.</p> <p><b>Height</b> was measured to the nearest 0.1 cm using the Holtain Harpenden stadiometer (Holtain Ltd, Crosswell, UK).</p> <p>Total <b>BF</b> (kg) was measured by DXA, and expressed as % total body weight.</p>	<p><b>%E (mean <math>\pm</math> SD)</b></p> <p><b>Free sugars</b>            Females: <math>4.1 \pm 3.8</math>            Males: <math>4.6 \pm 3.5</math></p> <p><b>Added sugars</b>            Females: <math>3.0 \pm 3.2</math>            Males: <math>3.6 \pm 3.0</math></p> <p><b>Method:</b> SFFQ</p>	<p><b>Free and added sugars</b> at baseline vs changes in body weight, BMI, BF and %BF over the 4-y follow-up</p> <p><b>Data collection:</b> baseline and end of follow-up</p>	<p><b>Model 1:</b> crude</p> <p><b>Model 2:</b> age, weight, history of CVD, monthly income, physical activity, education, smoking, and dietary intakes of whole grains, fruits and vegetables, red and processed meat, alcohol, green and Chinese tea, and caffeine</p>	<p><b>Significant positive</b> associations between the intake of added sugars and changes in BF and %BF in <b>males</b>. For each 1E% increase in added sugar intake, BF and %BF increased by 0.043 kg (<math>p=0.006</math>), and by 0.05% (<math>P=0.01</math>), respectively. Changes in body weight and BMI were in the <b>same direction (non-significant)</b>. Results for free sugars were similar. Only added sugar from beverages (35% of the total) significantly correlated with measures of body fatness.</p> <p>Non-significant (<b>positive</b>) associations for all these variables in females. Results were similar for free sugars.</p>
3	<b>KoCAS</b>  South Korea  Hur et al. (2015)  4 y  Public funding	<p><b>Same population and exclusion criteria as for total sugars</b></p>	<p><b>BMIZ score and %BF</b></p> <p><b>Same ascertainment of outcome as for total sugars</b></p>	<p><b>Baseline, free sugars from beverages g/d</b></p> <p><b>Median (IQR))</b>            0.4 (0.2, 2.4)</p> <p><b>Method:</b> 3-d DR</p>	<p><b>Free sugars</b> from beverages at baseline vs BMIZ scores and %BF at the end of the 4-y follow-up</p> <p><b>Data collection:</b> baseline and end of follow-up</p>	<p><b>Model 1:</b> crude</p> <p><b>Model 2:</b> total energy intake and household income at baseline. Sex and age only for %BF.</p>	<p>Associations between free sugars from beverages and BMIZ (<b>negative</b>) or %BF (<b>positive</b>) were <b>non-significant</b>. Per each 1 log (g/d) increase in free sugars from beverages at baseline, mean BMIZ was <math>-0.02</math> (<math>SE=0.03</math>) and %BF 0.02 (<math>SE=0.21</math>) in the most adjusted models.</p>
<b>Exposure: sucrose</b>							

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2	<b>EPIC-Norfolk</b>  UK  Kuhnle et al. (2015)  3 y  Public funding	<b>N</b> = 25,639  <b>Population sampled:</b> Norfolk's inhabitants  <b>Excluded:</b> Missing co-variables (i.e. sex, dietary data, second health check anthropometry), urinary sucrose analysis failed or outside the calibration range  <b>n</b> = 1,734 Females = 937 Male = 797  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 39 – 79 y	<b>BMI</b>  <b>Body weight and height</b> were measured at baseline and follow-up by trained research nurses using a standardised protocol.	<b>g/d † Geometric mean (SD)</b> Females: 45.0 (20.8) Males: 58.3 (29.1)  <b>g/MJ/d (range)</b> Females: 0.1 - 16.5 Males: 0.3 - 19.1  <b>% contribution to total sugars Geometric mean (SD)</b> Females: 43 (10) Males: 46 (12)  <b>Methods:</b> 24-h recall + 6-d DR = 7DD Urinary sucrose (spot urine)	<b>Sucrose</b> intake (7DD) and sucrose in urine at baseline vs BMI at the end of follow-up  <b>Data collection:</b> baseline for the exposure, baseline and end of follow-up for the outcome	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + physical activity	<b>Significant negative</b> associations between sucrose intake and BMI for males and females.  <b>7DD</b> <b>Per each 1 log(g/MJ/day) increase <math>\beta</math> coefficients (95% CI), kg/m<sup>2</sup></b>  <b>Females</b> Model 1: -1.60 (-2.25, -0.96) Model 2: -1.58 (-2.2, -0.93)  <b>Males</b> Model 1: -1.18 (-1.67, -0.69) Model 2: -1.18 (-1.68, -0.69)  <i>Associations between urinary sucrose and WC were in the opposite direction (positive, significant for females).</i>
2	<b>NSHDS</b>  Sweden  Winkvist et al. (2017)  10 y  Mixed funding	<b>N</b> = 40,066  <b>Population sampled:</b> General population  <b>Excluded:</b> Between visits interval <9y or >11y; >10% of FFQ missing or missing portion sizes; implausible energy intakes, missing body weight; weight < 35 kg, length <130 cm or BMI <15.  <b>n</b> = 15,995 Females = 8,354 Males = 7,641	<b>BMI</b>  Body <b>weight</b> and <b>height</b> were measured in light clothing without shoes, by trained nurses using standardized weight and measuring scales.	<b>E% Mean <math>\pm</math> SD</b> Females: 6.5 $\pm$ 2.6 Males: 6.6 $\pm$ 2.9  <b>Method:</b> SFFQ	Changes in <b>sucrose</b> intake vs changes in BMI over the 10-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> BMI, year of study participation, age, education, smoking status and physical activity at the beginning of the period  <b>Joint model</b> i.e. whole grain, PUFA, cholesterol, trans-fatty acids and sucrose entered in the same model	<b>Significant (females)</b> and non-significant (males) <b>negative</b> associations between changes in sucrose intake and changes in BMI over the follow-up  <b>Per each 1% change in E% <math>\beta</math> coefficients (SE), kg/m<sup>2</sup></b>  <b>Females:</b> -0.16 (0.07); p= 0.02 <b>Males:</b> -0.06 (0.04); p = 0.18



RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
		<b>Sex:</b> 52.2% Females <b>Ethnicity:</b> Caucasian <b>Age:</b> 30 – 60 y					
2	<b>PHHP</b> USA Parker et al. (1997) 4 y Public funding	<b>N</b> = 1,081 <b>Population sampled:</b> General population <b>Excluded:</b> Pregnancy, diabetes, missing BMI measurements, 10 or more missing items or extremely high or low scores for daily energy intake on the baseline FFQ <b>n</b> = 465 <b>Sex:</b> 62.2 % females <b>Ethnicity:</b> 94% Caucasian <b>Age:</b> 18 – 64 y	<b>Body weight</b> Body weight was measured by the interviewers with te participants in light clothing.	<b>g/d (range)†</b> <b>T1:</b> < 36.0 <b>T2:</b> 36.1-57.0 <b>T3:</b> >57.0 <b>Method:</b> SFFQ	<b>Sucrose</b> intake at baseline vs changes in body weight over the 4-follow-up	<b>Model:</b> age, BMI, smoking status, physical activity, total energy intake	Sucrose intake was not significantly associated with changes in body weight over the follow-up <b>Mean (SE) weight change (kg)</b> <b>T1:</b> 0.5 (0.5) <b>T2:</b> 1.3 (0.5) <b>T3:</b> 0.3 (0.6)
<b>Exposure: fructose</b>							
2	<b>SCES</b> Australia Gopinath et al. (2013) 5 y Mixed funding	<b>As for total sugars</b>	<b>BMI and %BF</b> <b>As for total sugars</b>	<b>Baseline, g/d †</b> <b>Females, NR</b> <b>Males (range)</b> <b>T1:</b> ≤26.1 <b>T2:</b> 26.2 – 34.6 <b>T3:</b> ≥34.7 <b>n</b> <b>T1:</b> 161 <b>T2:</b> 141 <b>T3:</b> 133 <b>Method:</b> SFFQ	<b>Females:</b> changes in <b>fructose</b> intake vs changes in BMI and %BF over follow-up <b>Males:</b> Intake of <b>fructose</b> at baseline vs changes in BMI and %BF over follow-up <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> age, ethnicity, parental education, passive smoking, change in energy intake, change in height, screen time and PAL	<b>Non-significant (positive)</b> associations between changes in fructose intake and changes in BMI or %BF during the 5-y follow-up after adjustment for confounders in females. For each SD increase in fructose (14.2g/d), mean BMI increased by 0.29 (SE = 0.16, p=0.07) and %BF by 0.46 (SE =0.40, p=0.25). In males, each SD increase in fructose at baseline (10.7 g/d) was associated with an increase in %BF of 0.52 (SE=NR, p=0.05). The association with BMI was also positive but non-significant (p=0.45).
<b>Exposure: SSSD / SSFD</b>							

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
2	<b>MTC</b>  Mexico  Stern et al. (2017)*  2 y  Unclear funding	<b>N</b> = 27,992  <b>Population sampled:</b> female teachers  <b>Excluded:</b> Diabetes, cancer, heart disease, ≥65 years, inadequate dietary information (energy intake <500 or >3500 kcal/day, response to ≤70 items in the dietary questionnaire, or missing cereal section), women with missing information on soda consumption in either 2006 or 2008. Women for whom BMI could not be calculated because of missing height or weight  <b>n</b> = 9,294  <b>Sex:</b> females  <b>Ethnicity:</b> Hispanic  <b>Age:</b> ≥25 y	<b>BW</b>  Participants self-reported <b>weight</b> (kg). Reproducibility and validity of self-reported anthropometry was evaluated in a subset of 3,413 participants. Standardized technician measurements were well correlated with self-reported weight ( $r = 0.92$ ). Changes in weight were calculated by subtracting self-reported measures in 2008 from those in 2006.	<b>Servings/d (mean ± SD)</b> 0.4 ± 0.5  <b>Change in servings/week from baseline (actual change; mean ± SD)</b> <b>G1:</b> < -1 (-3.7 ± 2.0) <b>G2 (ref):</b> -1 to 1 (-0.1 ± 0.4) <b>G3:</b> > 1 (2.8 ± 1.1)  <b>n</b> <b>G1:</b> 2,538 <b>G2:</b> 5,350 <b>G3:</b> 1,406  <b>Serving size:</b> 355 ml  <b>Method:</b> SFFQ	Change in <b>SSSD</b> intake vs changes in BW over the 2-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> baseline soda consumption (sugar and sugar-free), age, state (area), PAL, smoking, alcohol, changes in smoking and alcohol consumption, HRT, menopausal status, oral contraceptives, red meat, dairy, yogurt, fruit, nuts, vegetables, white bread, flour tortillas, corn tortillas, orange and grapefruit juice, homemade sweetened beverages	A <b>significant positive</b> relationship was observed between changes in SSSD intake and body weight changes over the 2-y follow-up. For each serving/day increase in SSSD intake, mean body weight increased by 1 kg (95% CI: 0.7, 1.2).  <b>β coefficients (95% CI) kg</b> <b>G1:</b> -0.4 (-0.6, -0.2) <b>G2 (ref):</b> 1 <b>G3:</b> 0.3 (0.2, 0.5)  <b>No relationship observed for ASSD</b>
2	<b>MIT-GDS</b>  USA  Phillips et al. (2004)  7 y (mean)  Mixed funding	<b>N</b> = 196  <b>Population sampled:</b> premenarcheal girls from Cambridge, MA  <b>Excluded:</b> incomplete or implausible dietary intake data, <3 annual visits, obesity defined as a triceps skinfold thickness >85th percentile for age and sex according to NHANES I, menarche.  <b>n</b> = 132 females	<b>BMIZ-score and %BF</b>  <b>Height</b> and body <b>weight</b> were measured in the morning. Height was measured to 0.1 cm with a wall-mounted stadiometer. Weight was measured with subjects in a hospital gown using a Seca scale accurate to 0.1 kg. <b>BMIZ-score</b> was calculated using the CDC modified growth reference standards.	<b>E%</b> <b>Q1 (ref):</b> <0.74 <b>Q2:</b> 0.75 to 1.4 <b>Q3:</b> 1.5 to 3.1 <b>Q4:</b> ≥ 3.2  <b>n per quartile</b> <b>NR</b>  <b>Method:</b> SFFQ	Intake of <b>SSSD</b> at baseline vs changes in BMIZ-score and %BF over the follow-up  <b>Data collection:</b> every year until 4 years after menarche (study exit).	<b>Model:</b> age at menarche, parental overweight, and servings of fruits and vegetables (for %BF: percentage of calories from protein)  *Other variables considered but not included in the model were physical activity index, inactivity time, race/ethnicity, percentage of daily calories from protein, carbohydrates, and fat (for	<b>Significant positive</b> association between baseline intake of SSSD and changes in BMIZ-score over the follow-up. The relationship with %BF was also <b>positive</b> , but non-significant.  <b>BMIZ-score</b> <b>β coefficients</b> <b>Q1:</b> ref <b>Q2:</b> 0.089 <b>Q3:</b> 0.172 <b>Q4:</b> 0.178 <b>P for trend</b> <0.001

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
		<b>Ethnicity:</b> 75% Caucasian, 14% Black, and 11% other  <b>Age:</b> 8 – 12 y	<b>%BF</b> was estimated by bioelectrical impedance analysis after an overnight fast or 2h postprandial. %BF was estimated using prediction equations developed in this cohort, with measures of total body water by isotopic dilution of H <sub>2</sub> <sup>18</sup> O as the criterion method. Separate equations were used depending on the menarcheal status of the participant  Visits every year until 4 years after menarche			%BF also servings of fruits and vegetables)	<b>%BF</b> <b>β coefficients</b> <b>Q1:</b> ref <b>Q2:</b> 0.15 <b>Q3:</b> 0.41 <b>Q4:</b> 0.31 <b>P for trend = 0.23</b>
2	<b>NGHS</b>  USA  Striegel-Moore et al. (2006)  10 y  Unclear funding	<b>N</b> = 2,379  <b>Population sampled:</b> Non-Hispanic Caucasian and African American girls with racially concordant parents from 3 sites  <b>Excluded:</b> not having at least one 3-d DR  <b>Follow-up rate</b> @ 90% <b>n</b> = 2,371  <b>Sex:</b> females <b>Ethnicity:</b> 51% Black, 49% Caucasian <b>Age:</b> 9 – 10 y	<b>BMI</b>  <b>Weight</b> and <b>height</b> were measured annually by research staff.	<b>g/d (mean (SE))</b> NR for pooled cohort  <b>SSSD</b> , Caucasian <b>v1:</b> 135.45 (8.29) <b>v10:</b> 377.02 (9.09) <b>SSSD</b> , Black <b>v1:</b> 134.53 (7.85) <b>v10:</b> 338.48 (8.11)  <b>SSFD</b> , Caucasian <b>v1:</b> 78.41 (4.39) <b>v10:</b> 87.16 (9.09) <b>SSFD</b> , Black <b>v1:</b> 134.68 (4.86) <b>v10:</b> 204.41 (7.00)  <b>Method:</b> 3-d DR	1-y change in <b>SSSD and SSFD</b> intake vs 1-y change in BMI  <b>Data collection:</b> every year. Each observation refers to two consecutive years.	<b>Model:</b> site, visit, race, total energy intake and consumption of milk, ASSD, fruit juice, coffee/tea and SSFD (for analysis of SSSD) or SSFD (for analysis of SSFD)	<b>Positive</b> associations between 1-y change in intake of <b>SSSD (significant)</b> and SSFD (non-significant) and 1-y change in BMI  <b>Per each 100 g/d increase β coefficients (SE), kg/m2</b>  <b>SSSD</b> 0.011 (0.005), <b>P &lt; 0.05</b>  <b>SSFD</b> 0.009 (0.007), <b>NS</b>  <i>Relationship for ASSD was negative and non-significant.</i>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
3	<b>DCH</b>  Denmark  Olsen et al. (2016)  5 y  Mixed funding	<b>N</b> = 57,053  <b>Population sampled:</b> Inhabitants from Copenhagen and Aarhus counties  <b>Excluded:</b> If aged >60 y at baseline and aged >65 y at follow-up, history of cancer or developed cancer, CVD, or diabetes during the study period, had unstable smoking habits between baseline and follow-up, and had a mean gain in BW >5 kg/y.  <b>n</b> = 2,165 <b>Sex:</b> 49.4% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 50 – 64 y	<b>BW</b>  Baseline <b>BW</b> was measured to the nearest 0.1 kg by project staff. Follow-up measures of BW were self-reported.	<b>ml/d median (95% CI)</b> 10.5 (0.3, 200.3)  <b>Method:</b> SFFQ	Intake of <b>SSSD</b> at baseline vs annual changes in BW and over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> baseline weight, height, sex, age, smoking status, alcohol consumption, PAL, education, menopausal status  <b>Model 2:</b> model 1 + energy intake	<b>Significant positive</b> association between intake of SSSD at baseline and annual changes in BW over the follow-up.  <b>Per 200 ml/d increase <math>\beta</math> coefficients (95% CI) kg/y</b> <u>Model 1:</u> 0.10 (0.01, 0.18) <u>Model 2:</u> 0.12 (0.03, 0.20)
3	<b>Inter99</b>  Denmark  Olsen et al. (2016)  2 y  Mixed funding	<b>N</b> = 13,016  <b>Population sampled:</b> Inhabitants from Copenhagen county  <b>Excluded:</b> Prevalent cancer, CVD, or self-reported diabetes at baseline or had incident cancer, CVD or self-reported diabetes during follow-up.  <b>n</b> = 1,341 <b>Sex:</b> 49.3% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 30 – 60 y	<b>BW</b>  Baseline and follow-up <b>BW</b> was measured to the nearest 0.1 kg by project staff.	<b>ml/d median (95% CI)</b> 16.4 (0, 500)  <b>Method:</b> SFFQ	Intake of <b>SSSD</b> at baseline vs annual changes in BW and over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> baseline weight, height, sex, age, smoking status, alcohol consumption, PAL, education, menopausal status  <b>Model 2:</b> mode 1 + energy intake	<b>Negative (non-significant)</b> association between intake of SSSD at baseline and annual changes in BW over the follow-up.  <b>Per 200 ml/d increase <math>\beta</math> coefficients (95% CI) kg/y</b> <u>Model 1:</u> -0.03 (-0.19, 0.13) <u>Model 2:</u> -0.02 (-0.19, 0.15)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
3	<b>MONICA</b>  Denmark  Olsen et al. (2016)  5 y  Public funding	<b>N</b> = 4,581  <b>Population sampled:</b> Inhabitants from Copenhagen county  <b>Excluded:</b> Prevalent cancer, CVD, or self-reported diabetes at baseline or had incident cancer, CVD or self-reported diabetes during follow-up.  <b>n</b> = 1,257 <b>Sex:</b> 52.1% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 30 – 60 y	<b>BW</b>  Baseline and follow-up <b>BW</b> was measured to the nearest 0.1 kg by project staff.	<b>ml/d median (95% CI)</b> 0 (0, 250)  <b>Method:</b> 7-d DR	Intake of <b>SSSD</b> at baseline vs annual changes in BW and over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> baseline weight, height, sex, age, smoking status, alcohol consumption, PAL, education, menopausal status  <b>Model 2:</b> model 1 + energy intake	<b>Positive (non-significant)</b> association between intake of SSSD at baseline and annual changes in BW over the follow-up.  <b>Per 200 ml/d increase <math>\beta</math> coefficients (95% CI) kg/y</b> <u>Model 1:</u> 0.04 (-0.06, 0.14) <u>Model 2:</u> 0.05 (-0.05, 0.14)
3	<b>GUTS II</b>  USA  Field et al. (2014)*  7 y  Public funding	<b>N</b> = 10,919  <b>Population sampled:</b> offspring of participants from NHSII  <b>Excluded:</b> Missing data on vigorous activity or reporting more than 40 hours per week (outliers). Missing data or outliers (>70 h per week) on time spent watching TV and missing data on sports drink or diet soda consumption.  <b>n</b> = 7,559 Females = 4,121 Males = 3,438  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 9 – 15 y	<b>BMI</b>  <b>BMI</b> (kg/m <sup>2</sup> ) was calculated using self-reported <b>weight</b> and <b>height</b> . <b>Change in BMI</b> was modeled as BMI at the end of the time interval, controlling for BMI at the beginning of the time interval and time between assessments. Participants contributed with information on BMI change during up to three time periods: 2004-2006, 2006-2008, and/or 2008-2011.	<b>Servings/d NR</b>  <b>Serving size:</b> 355 ml  <b>Method:</b> SFFQ	Intake of <b>SSSD</b> at the beginning of each 2-3 y time period and change in <b>SSSD</b> intake over each 2-3 y time period vs change in BMI over the same 2-3 y time period  <b>Data collection:</b> baseline, 2 and 4 y later and end of follow-up	<b>Model 1:</b> age, time between questionnaires, BMI at the start of the time period, diet soda intake, sport drink intake  <b>Model 2:</b> model 1 + hours per day of TV watching, hours per week of vigorous activity  <b>Model 3:</b> model 1 + soda intake at the start of the time period  <b>Model 4:</b> model 2 + soda intake at the start of the time period	<b>Positive</b> (non-significant) association between intake of SSSD at the beginning of each period and change in BMI over the each 2-3 y time period for both sexes. The association was also positive for sport drinks and significant in females. The association between change in SSSD intake and concurrent change in BMI over the same 2-3 y time period was <b>positive</b> (non-significant) for both sexes. The association was also positive for <b>sport drinks</b> and significant in males.  <b>Exposure: Baseline</b>  <b>Per each 1 serving/day increase <math>\beta</math> coefficients (95% CI) kg/m<sup>2</sup></b>  <b>Females</b>

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							<p><u>Model 1</u>: -0.20 (-0.12, 0.08) <u>Model 2</u>: 0.00 (-0.10, 0.10)</p> <p><b>Males</b> <u>Model 1</u>: 0.05 (-0.05, 0.15) <u>Model 2</u>: 0.05 (-0.06, 0.16)</p> <p><b><i>Positive relationship observed for ASSD (significant in females only)</i></b></p> <p><b><u>Exposure: 2-3y change</u></b></p> <p><b>Per each 1 serving/d increase <math>\beta</math> coefficients (95% CI) kg/m<sup>2</sup></b></p> <p><b>Females</b> <u>Model 1</u>: 0.09 (-0.03, 0.21) <u>Model 2</u>: 0.10 (-0.03, 0.22) <u>Model 3</u>: 0.11 (-0.06, 0.27) <u>Model 4</u>: 0.12 (-0.05, 0.29)</p> <p><b>Males</b> <u>Model 1</u>: 0.09 (-0.04, 0.22) <u>Model 2</u>: 0.08 (-0.06, 0.22) <u>Model 3</u>: 0.15 (0.00, 0.30) <u>Model 4</u>: 0.14 (-0.02, 0.30)</p> <p><b><i>Positive relationship observed for ASSD (significant in males only)</i></b></p>
<b>Exposure: SSSD+SSFD</b>							

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
1	<b>ALSPAC</b>  UK  Johnson et al. (2007)  4.6 y (mean)  Mixed funding	<b>N</b> = 14,541  <b>Population sampled:</b> General population living within a defined part of the country  <b>Excluded:</b> Women who were resident in Avon while pregnant but left shortly after enrolment were omitted from further follow-up.  <b>n</b> = 521 (model 2 = 362)  <b>Sex:</b> mixed, females proportion NR <b>Ethnicity:</b> Caucasian <b>Age:</b> 5 y	<b>BF (kg)</b>  <b>BF</b> was measured by DXA.	<b>g/d</b> <b>Median (IQR)</b> Age 5 y: 57 (0, 163) Age 7 y: 67 (0, 196)  <b>Serving size:</b> 180 ml  <b>Method:</b> 3-d DR	Intake of <b>SSSD+SSFD</b> at 5 and 7 y vs BF at 9 y (end of follow-up)  <b>Data collection:</b> dietary data at 5 and 7 y, BF at 9 y (end of follow-up).	<b>Model 1:</b> sex, height at outcome assessment  <b>Model 2:</b> model 1 + baseline BMI, TV watching, maternal education, paternal class, maternal BMI, paternal BMI, misreporting of energy intake, dietary energy density, %E from fat, fibre density	Non-significant <b>negative</b> associations between intake of SSSD+SSFD at 5 and 7 y and BF at 9 y.  <b>Per each 1 serving/day increase</b> <b><math>\beta</math> coefficients (95% CI), kg</b>  <b>5 y</b> <u>Model 1:</u> -0.16 (-0.60, 0.28) <u>Model 2:</u> -0.15 (-0.54, 0.24)  <b>7 y</b> <u>Model 1:</u> -0.13 (-0.47, 0.22) <u>Model 2:</u> -0.11 (-0.37, 0.15)
1	<b>ALSPAC</b>  UK  Bigornia et al. (2015)  3 y (mean)  Mixed funding	<b>N</b> = 14,541  <b>Population sampled:</b> General population living within a defined part of the country  <b>Excluded:</b> missing anthropometric, DXA, dietary and/or physical activity information  <b>n</b> = 2,455 (model 4 = 1,059)  <b>Sex:</b> 53.0% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 10 y	<b>BW, BMI and BF (kg)</b>  <b>Weight</b> was measured in kilograms using a Tanita body fat analyser and <b>height</b> in millimetres using a Harpenden stadiometer at baseline and follow-up.  <b>BF</b> was measured by DXA.	<b>Servings/d (median (IQR))</b> Females: 0.3 (1.0) Males: 0.4 (1.4)  <b>Change in servings/day from baseline (mean (SD))</b> 0.12 (1.36)  <b>Serving size:</b> 180 ml  <b>Method:</b> 3-d DR	Change in <b>SSSD+SSFD</b> intake vs change in BW, BMI, and BF over the 3-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> change in SSSD+SSFD intake from baseline, baseline SSSD+SSFD intake, sex, and baseline age, height and baseline adiposity (for BF, BMI was used, for others not defined)  <b>Model 2:</b> model 1 + PAL at 13 y, pubertal stage at 13 y, maternal overweight/obesity, maternal education, dieting at 13 y, change from baseline in fruit juice, fruit, vegetable and fat intake  <b>Model 3:</b> model 2 + dietary reporting errors at 13 y	<b>Significant positive</b> associations between change in intake of SSSD+SSFD and change in BW, BMI and BF over the 3-y follow-up after accounting for dietary misreporting. Associations were attenuated (BW by 47%, BMI by 25%, BF not affected) when adjusting for total energy in sensitivity analyses and were independent from baseline consumption of SSSD+SSFD  <b>Per each 1 serving/day increase</b> <b>BMI, <math>\beta</math> coefficients (SE), kg/m<sup>2</sup></b> <u>Model 1:</u> 0.07 (0.03), P = 0.023 <u>Model 2:</u> 0.07 (0.03), P = 0.025 <u>Model 3:</u> 0.09 (0.03), P = 0.002 <u>Model 4:</u> 0.16 (0.04), p < 0.001

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						<b>Model 4:</b> model 2 among plausible dietary reporters at 13 y	<b>BF, <math>\beta</math> coefficients (SE), kg</b> <b>Model 1:</b> 0.08 (0.08), P = 0.298 <b>Model 2:</b> 0.10 (0.08), P = 0.203 <b>Model 3:</b> 0.19 (0.08), P = 0.011 <b>Model 4:</b> 0.33 (0.11), P = 0.003
<b>1</b>	<b>Framingham-3Gen</b>  USA  Ma et al. (2016b)b  6 y  Public funding	<b>N</b> = 4,095  <b>Population sampled:</b> General population/third generation of the Framingham Heart Study  <b>Excluded:</b> Not eligible for CT scans (BW >160 kg, women <40 y, men <35 y), missing CT scan at baseline or follow-up, missing data on exposure or covariates, bariatric surgery, history of CVD or cancer  <b>n</b> = 1,003  <b>Sex:</b> 53.3% females  <b>Ethnicity:</b> 99.7% Caucasian  <b>Age:</b> 19 – 72 y	<b>BW</b>  <b>BW</b> was measured with light clothes, and was rounded to the nearest 0.5 pound	<b>Servings/week Range (median)</b> <b>G1:</b> 0 – < 0.25 (0) <b>G2:</b> 0.25 – < 1 (0.5) <b>G3:</b> 1 – <7 (3) <b>G4:</b> >7 (11)  <b>Serving size</b> = 12 oz (355mL)  <b>n</b> <b>G1:</b> 317 <b>G2:</b> 196 <b>G3:</b> 356 <b>G4:</b> 134  <b>Method:</b> SFFQ	Intake of <b>SSSD+SSFD</b> at baseline vs changes in BW over the 6-y follow-up  <b>Data collection:</b> exposure at baseline and outcome at baseline and end of follow-up	<b>Model:</b> baseline weight, sex, age, smoking status, physical activity score, energy intake (kcal/day), alcohol intake (g/d), saturated fat intake (%energy), diet soda intake (servings/week), multivitamin use, whole grain, fruit, vegetable, coffee (servings/day), nuts and fish	<b>Negative (non-significant)</b> association between baseline intake of SSSD+SSFD and change in BW over the follow-up.  <b>Change in BW (kg) Mean (95% CI)</b> <b>G1:</b> 2.4 (1.7, 3.2) <b>G2:</b> 2.8 (1.8, 3.7) <b>G3:</b> 2.4 (1.7, 3.0) <b>G4:</b> 1.7 (0.5, 2.9) <b>P for trend</b> = 0.26  <b>No relationship observed for ASSD</b>



RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
1	<b>SUN</b>  Spain  Barrio-Lopez et al. (2013)  6 y  Public funding	<b>N</b> = 14,716  <b>Population sampled:</b> University graduates, mainly health professionals  <b>Excluded:</b> Having one or more criteria for MetS, extreme energy intake (<800 or >4000 kcal/d for men and <500 and >3500 kcal for women), not answering the 6-year or 8-year follow-up questionnaire.  <b>n</b> = 8,157  <b>Sex:</b> 69% females  <b>Ethnicity:</b> Caucasian  <b>Age (mean):</b> 36 y	<b>BW</b>  <b>Weight</b> was self-reported through questionnaires. The validity of self-reported weight in this study has been previously assessed and the correlation coefficient between self-reported and measured weight was 0.991 (95% CI:0.986 to 0.994) <sup>5</sup> .	<b>Baseline (ml/d)</b> <b>Mean ± SD</b> Q1: 109.6 ± 119.8 Q2: 26.53 ± 35.1 Q3: 0 Q4: 13.5 ± 9.9 Q5: 58.6 ± 80.6  <b>Change in consumption</b> <b>Range (ml/d)</b> Q1 (ref): ≤-28.57 Q2: -28.58 – <0 Q3: 0 Q4: >0 – 33.81 Q5: >33.81  <b>Median</b> <b>(servings/week)</b> Q1: -1.35 Q2: -0.3 Q3: 0 Q4: 0.4 Q5: 2.4  <b>Serving size</b> = 330 ml  <b>n</b> Q1: 1,890 Q2: 1,334 Q3: 1,796 Q4: 1,626 Q5: 1,511  <b>Method:</b> SFFQ	Change in <b>SSSD+SSFD</b> intake vs change in body weight over the follow-up  <b>Data collection:</b> exposure at baseline and end of follow-up and outcome every 2 years	<b>Model 1:</b> crude  <b>Model 2:</b> age, sex  <b>Model 3:</b> model 2 + baseline BMI, smoking, PAL, alcohol intake, soft drink intake at baseline, total energy intake, red meat, french fries, fast food consumption, Mediterranean diet pattern	<b>A significant positive</b> relationship was observed between changes in SSSD intake and body weight changes over the 6-y follow-up. The highest quantile of increase in SSSD consumption (median = + 2.4 servings/week) gained an average of 1.3 kg (95 % CI 1.1, 1.6) more than the lowest quintile, where consumptions of SSSD was reduced (median = - 1.35 servings/week).  <b>Per quintile of change in intake</b> <b>β coefficients (95% CI)</b> <b>Model 1</b> Q1 (ref): 0 Q2: 2.6 (1.7, 4.0) Q3: 3.5 (2.3, 5.2) Q4: 3.0 (3.0, 4.6) Q5: 3.2 (2.1, 4.8) <b>P for trend &lt;0.001</b>  <b>Model 2</b> Q1 (ref): 0 Q2: 0.4 (0.1, 0.7) Q3: 0.3 (0.0, 0.6) Q4: 0.9 (0.6, 1.2) Q5: 1.3 (1.0, 1.6) <b>P for trend &lt;0.001</b>  <b>Model 3</b> Q1 (ref): 0 Q2: 0.5 (0.2, 0.8) Q3: 0.5 (0.2, 0.8)

<sup>5</sup> Bes-Rastrollo M, Pérez Valdivieso JR, Sánchez-Villegas A, et al. Validación del peso e índice de masa corporal auto-declarados de los participantes de una cohorte de graduados universitarios. Rev Esp Obes. 2005; 3:183-9.

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							Q4: 1.1 (0.8, 1.4) Q5: 1.3 (1.1, 1.6) <b>P for trend &lt;0.001</b>
1	<b>HPFS</b>  USA  Pan et al. (2013)  20 y  Public funding	<b>N</b> = 51,529  <b>Population sampled:</b> male health professionals  <b>Excluded:</b> Missing data on body weight, beverages, lifestyle habits, > 9 blank responses on the baseline dietary questionnaire, implausible energy intakes (<900 or >3,500 kcal/d), age >65 y; diabetes, cancer, cardiovascular, pulmonary, renal, or liver disease at baseline.  <b>n</b> = 21,988 <b>Sex:</b> males <b>Ethnicity:</b> Ethnicity: Caucasian (~90%+) <b>Age:</b> 40 – 75 y	<b>BW</b>  <b>Weight</b> was self-reported and assessed every 2 years through questionnaires.	<b>servings/d</b> <b>Mean (95% CI)</b> 0.37 (0, 1.36)  <b>Serving size:</b> 355 ml  <b>Method:</b> SFFQ	Change in <b>SSSD+SSFD</b> intake vs change in BW within each 4-y interval over the follow-up  <b>Data collection:</b> every 4 years during follow-up	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + baseline BMI, sleep duration, changes in PAL, alcohol use, TV watching, smoking, other beverages, dietary variables (fruits, vegetables, whole grain, refined grain, potatoes, potato chips, red meat, other dairy products, sweets and desserts, nuts, fried foods and trans-fat)	<b>Significant positive</b> relationship between change in SSSD+SSFD intake and change in BW within each 4-y interval over the follow-up.  <b>Per each 1 serving/day increase</b> <b><math>\beta</math> coefficients (95% CI), kg</b> <b>Model 1:</b> 0.38 (0.31, 0.44) <b>Model 2:</b> 0.25 (0.19, 0.31)  <b>Significant inverse relationship observed for ASB</b>
1	<b>NHS</b>  USA  Pan et al. (2013)  20 y	<b>N</b> = 121,700  <b>Population sampled:</b> female nurses  <b>Excluded:</b> Missing data on body weight, beverages, lifestyle habits, > 9 blank responses on the	<b>BW</b>  <b>Weight</b> was self-reported and assessed every 2 years through questionnaires. In a validation study among 184 women from the NHS, participants were weighed 6 to	<b>servings/d</b> <b>Mean (95% CI)</b> 0.24 (0, 1.07)  <b>Serving size:</b> 355 ml  <b>Method:</b> SFFQ	Change in <b>SSSD+SSFD</b> intake vs change in BW within each 4-y interval over the follow-up	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + baseline BMI, sleep duration, changes in PAL, alcohol use, TV watching, smoking, other beverages, dietary variables (fruits, vegetables, whole	<b>Significant positive</b> relationship between change in SSSD+SSFD intake and change in BW within each 4-y interval over the follow-up  <b>Per each 1 serving/day increase</b>

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	Public funding	baseline dietary questionnaire, implausible energy intakes (<900 or >3,500 kcal/d), age >65 y; diabetes, cancer, cardiovascular, pulmonary, renal, or liver disease at baseline, pregnancy at follow-up.  <b>n</b> = 50,013 <b>Sex:</b> females <b>Ethnicity:</b> Caucasian <b>Age:</b> 30 – 55 y	12 months after completing the mailed questionnaire. Reported weights were highly correlated with measured weights (Spearman correlation coefficient = 0.96), although they averaged 1.5 kg (3.3 lb) lower than the measured values <sup>6</sup> .		<b>Data collection:</b> every 4 years during follow-up	grain, refined grain, potatoes, potato chips, red meat, other dairy products, sweets and desserts, nuts, fried foods and trans-fat)	<b>β coefficients (95% CI), kg</b> <b>Model 1:</b> 0.50 (0.44, 0.54) <b>Model 2:</b> 0.36 (0.30, 0.41)  <b>Significant inverse relationship observed for ASB</b>
<b>1</b>	<b>NHS II</b>  USA  Pan et al. (2013)  16 y  Public funding	<b>N</b> = 116,671  <b>Population sampled:</b> female nurses  <b>Excluded:</b> Missing data on body weight, beverages, lifestyle habits, > 9 blank responses on the baseline dietary questionnaire, implausible energy intakes (<900 or >3,500 kcal/d), age >65 y; diabetes, cancer, cardiovascular, pulmonary, renal, or liver disease at baseline, pregnancy at follow-up.  <b>n</b> = 52,987 <b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 25 – 42 y	<b>BW</b>  <b>Weight</b> was self-reported and assessed every 2 years through questionnaires. In a validation study among 184 women from the NHS, participants were weighed 6 to 12 months after completing the mailed questionnaire. Reported weights were highly correlated with measured weights (Spearman correlation coefficient = 0.96), although they averaged 1.5 kg (3.3 lb) lower than the measured values <sup>7</sup> .	<b>servings/d</b> <b>Mean (95% CI)</b> 0.46 (0, 2.5)  <b>Serving size:</b> 355 ml  <b>Method:</b> SFFQ	Change in <b>SSSD+SSFD</b> intake vs change in BW within each 4-y interval over the follow-up  <b>Data collection:</b> every 4 years during follow-up	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + baseline BMI, sleep duration, changes in PAL, alcohol use, TV watching, smoking, other beverages, dietary variables (fruits, vegetables, whole grain, refined grain, potatoes, potato chips, red meat, other dairy products, sweets and desserts, nuts, fried foods and trans-fat)	<b>Significant positive</b> relationship between change in SSSD+SSFD intake and change in BW within each 4-y interval over the follow-up.  <b>Per each 1 serving/day increase</b> <b>β coefficients (95% CI), kg</b> <b>Model 1:</b> 0.66 (0.61, 0.70) <b>Model 2:</b> 0.47 (0.42, 0.52)  <b>Significant inverse relationship observed for ASB</b>

<sup>6</sup> Manson JE, Willett WC, Stampfer MJ, Colditz GA, Hunter DJ, Hankinson SE, et al. Body weight and mortality among women. N Engl J Med. 1995; 333:677–685. [PubMed: 7637744]

<sup>7</sup> Manson JE, Willett WC, Stampfer MJ, Colditz GA, Hunter DJ, Hankinson SE, et al. Body weight and mortality among women. N Engl J Med. 1995; 333:677–685. [PubMed: 7637744]

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2	<b>CoSCIS</b>  Denmark  Jensen et al. (2013)  7 y  Mixed funding	<b>N</b> = 1,024  <b>Population sampled:</b> children entering a public school in two suburbs of Copenhagen  <b>Excluded:</b> Incomplete dietary records, extreme intake of sweet drinks (>1400 g/d), missing information on beverage intake, BMI $\Sigma$ 4SF or SES.  <b>n</b> = 286  <b>Sex:</b> 51.1% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 6 y (mean)	<b>BMI and SFT</b>  <b>Weight and height</b> were measured without shoes and with light indoors clothing to nearest 0.1 kg using a calibrated beam balance and to nearest 0.1 cm using a stadiometer respectively. <b>BMI</b> was calculated as weight (kg)/height (m) <sup>2</sup> .  <b>Skin-fold thicknesses</b> (mm) (SFT) were measured with Harpenden callipers at four points on the non-dominant side of the body: (i) triceps; (ii) biceps; (iii) subscapularly and (iv) supra iliaca (31). The variable, $\Sigma$ 4SF, was generated by summarizing the four measurements.	<b>SSSD+SSFD combined NR</b>  <b>SSSD g/d Median (IQR)</b> 114 (57, 200)  <b>SSFD g/d Median (IQR)</b> 143 (46, 267)  1 g ~ 1 ml  <b>Method:</b> 7-d DR	Intake of <b>SSSD+SSFD</b> at baseline vs changes in BMI and $\Sigma$ 4SF over the follow-up  <b>Data collection:</b> exposure at baseline and 3 y later and outcome at baseline, 3 y later and end of follow-up	<b>Model:</b> baseline BMI (log $\Sigma$ 4SF for SFT), school cluster, sex, SES and intervention/comparison group.	Each 100 ml/d increase of SSSD+SSFD intake at baseline was <b>negatively (non-significant)</b> associated with a change in BMI of -0.059 kg/m <sup>2</sup> (95% CI: -0.145, 0.027) and in log $\Sigma$ 4SF of -0.004 mm (95% CI: -0.019, 0.010) over the 7 y follow-up.
2	<b>MOVE</b>  USA  Carlson et al. (2012)  2 y  Public funding	<b>N</b> = 271  <b>Population sampled:</b> Children with history of parental obesity  <b>Excluded:</b> Living in a foster or group home, having a medical and/or psychological condition affecting diet, physical activity, growth, or weight, being unable to speak, read, and understand either English or Spanish.  <b>Follow-up rate:</b> 94.8%	<b>BMIZ-score and %BF</b>  <b>Weight and height</b> were measured by trained staff, and <b>BMIZ-scores</b> for age and gender were calculated using CDC growth charts. <b>Body fat percentage</b> was estimated by bioelectrical impedance analysis and using the Schaefer equation for children of this age <sup>8</sup> . A validation study (n=30) showed high correlation	<b>Servings/d Mean <math>\pm</math> SD</b> 0.54 $\pm$ 0.59  <b>Serving size</b> = 355 ml  <b>Method:</b> SFFQ	Change in <b>SSSD+SSFD</b> intake vs change in BMIZ-score and %BF over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> age, gender, ethnicity, parent education, and height	<b>Positive</b> association between change in SSSD+SSFD and changes in BMIZ-score ( <b>non-significant</b> ) and %BF ( <b>significant</b> ) over the 2 y follow-up.  <b>Per each serving/d increase <math>\beta</math> coefficient (95% CI)</b>  <b>BMIZ-score</b> 0.11 (-0.03, 0.25), P = 0.124  <b>%BF</b> 1.40 (0.09, 2.72), <b>P = 0.036</b>

<sup>8</sup> Schaefer F, Georgi M, Zieger A, et al. Usefulness of bioelectric impedance and skinfold measurements in predicting fat-free mass derived from total body potassium in children. *Pediatr Res* 1994;35:617-624.

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		<b>n</b> = 254 <b>Sex:</b> 56% females <b>Ethnicity:</b> 39% Caucasian, 48% Latino, 13% other <b>Age:</b> 6-7 y	with DXA measured percent body fat ( $r = 0.84$ )				
3	<b>GUTS</b>  USA  Berkey et al. (2004)  2 y  Mixed funding	<b>N</b> = 16,771  <b>Population sampled:</b> offspring of participants from NHSII  <b>Excluded:</b> implausible energy intakes, height that was >3 SD beyond the gender-age-specific mean height, any 1-year height change which declined by >1 inch or increased by >3 SD above the mean change, BMI < 12 kg/m <sup>2</sup> and BMI < 3 SD above or below the gender-age-specific mean  <b>n</b> = 11,755 Females = 6,688 Males = 5,067  <b>Ethnicity:</b> 94.7% Caucasian, 5.3% other  <b>Age:</b> 9 – 14 y	<b>BMI</b>  <b>Weight</b> and <b>height</b> were self-reported by the children in the annual questionnaire. They were provided specific measurement instructions and suggested to ask someone for help.	<b>Serving/d</b> NR for cohort combined  <b>Serving size</b> = 355 ml  <b>Method:</b> SFFQ	Intake of <b>SSSD+SSFD</b> at baseline and 1-y change in <b>SSSD+SSFD</b> intake vs 1-y change in BMI  <b>Data collection:</b> baseline, and 1 and 2 years of follow-up	<b>Model 1:</b> age, Tanner stage, race, menarche (girls), prior BMI z score, height, milk type, physical activity, inactivity and baseline beverage intake  <b>Model 2:</b> model 1 + total energy intake	<b>Positive (non-significant)</b> associations between baseline intake and 1-y change in intake of SSSD+SSFD and 1-y change in BMI.  <b>Exposure: baseline</b> <b>Per each serving/d increase <math>\beta</math> coefficients (SE) kg/m<sup>2</sup>/y</b> <b>Females</b> Model 1: 0.021 (0.012), $p = 0.096$ Model 2: 0.019 (0.014), $p = 0.167$ <b>Males</b> Model 1: 0.028 (0.014), $p = 0.038$ Model 2: 0.015 (0.015), $p = 0.317$  <b>Positive relationship observed for ASSD (significant for males only)</b>  <b>Exposure: 1-y change</b> <b>Per each serving/d increase <math>\beta</math> coefficients (SE) kg/m<sup>2</sup>/y</b>

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							<b>Females</b> Model 1: 0.026 (0.015), <b>p = 0.082</b> Model 2: 0.023 (0.016), <b>p = 0.159</b> <b>Males</b> Model 1: 0.040 (0.016), <b>p = 0.012</b> Model 2: 0.024 (0.018), <b>p = 0.178</b>  <i>Similar relationship observed for ASSD</i>
<b>Exposure: SSSD+SSFD+SSFJ</b>							
<b>1</b>	<b>WAPCS</b> Australia Ambrosini et al. (2013) 3 y Unclear funding	<b>N = 2,868</b>  <b>Population sampled:</b> offspring from mothers from the Raine study  <b>Excluded:</b> Subjects who reported not fasting before venepuncture.  <b>n = 1,366</b> Females = 660 Males = 706 <b>Sex:</b> 48.3% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 14 y	<b>BMI</b>  Calibrated measurements of <b>height</b> and <b>weight</b> were made by using electronic chair scales and a stadiometer.	<b>g/d mean ± SD (range)</b> T1 (ref): 48 ± 39 (0 – 130) T2: 223 ± 59 (130 – 329) T3: 665 ± 351 (331 – 2,876)  <b>n of those who changed tertiles between 14 and 17 y NR</b>  <b>Method:</b> SFFQ	Changes in <b>SSSD+SSFD+SSFJ</b> intake vs percent of change in BMI over the 3-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> age, pubertal stage, physical fitness, dietary misreporting, maternal education, family income  <b>Model 2:</b> model 1 + healthy and Western diet pattern scores	<b>Significant positive</b> association between changes in SSSD+SSFD+SSFJ intake and changes in BMI over the 3-y follow-up, in females, but not males.  <b>Per each tertile of intake increase Δ% (95% CI) vs T1</b>  <b>Females</b> <b>Model 1:</b> T2: 0.5 (-1.2, 2.2) T3: 3.8 (1.8, 5.7) <b>P for trend &lt;0.001</b>  <b>Model 2:</b> T2: 0.4 (-1.3, 2.1) T3: 3.6 (1.5, 5.8) <b>P for trend = 0.002</b>  <b>Males</b> <b>Model 1:</b> T2: 0.6 (1.3, 2.1)

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							<p><u>T3</u>: 1.5 (-0.5, 3.5) <b>P for trend = 0.14</b></p> <p><b>Model 2:</b> <u>T2</u>: 0.3 (-1.6, 2.3) <u>T3</u>: 0.8 (-1.3, 2.9) <b>P for trend = 0.46</b></p>
1	<b>WHI</b>  USA  Auerbach et al. (2018)  3 y  Public funding	<p><b>N</b> = 122,970</p> <p><b>Population sampled:</b> Postmenopausal women recruited from 40 clinical centres</p> <p><b>Excluded:</b> Missing baseline and year 3 body weight or 100% FJ intake, baseline age &gt;65 y, BMI &gt; 35.0 kg/m<sup>2</sup> and implausible energy intake</p> <p><b>n</b> = 49,106 <b>Sex:</b> females <b>Ethnicity:</b> 84% Caucasian, 7.6% Black, Hispanic/Latino 4% and 3% Asian/Pacific <b>Age:</b> 50 – 65 y</p>	<p><b>BW</b></p> <p>Study personnel measured <b>BW</b> using a standardized protocol and calibrated scales.</p>	<p><b>Servings/d<sup>+</sup></b> 0.30 ± 0.54</p> <p><b>Serving size:</b> 6oz (177 ml)</p> <p><b>Method:</b> SFFQ</p>	<p>Change in <b>SSSD+SSFD+SSFJ</b> vs change in BW (lbs) over the 3-y follow-up</p> <p><b>Data collection:</b> baseline and end of follow-up</p>	<p><b>Model 1:</b> crude</p> <p><b>Model 2:</b> age, education, income, ethnicity, current smoking, BMI, HRT, PAL, change in healthy eating index diet quality score</p> <p><b>Model 3:</b> model 2 + change in total energy intake</p>	<p><b>Significant positive</b> association between change in SSSD+SSFD+SSFJ intake and change in BW (lbs) over the 3-y follow-up.</p> <p><b>Per each 1 serving/day increase</b> <b>β coefficients (95% CI), lbs</b> <u>Model 1</u>: 0.93 (0.62, 1.24) <u>Model 2</u>: 0.58 (0.26, 0.90) <u>Model 3</u>: 0.36 (0.29, 0.69)</p>
1	<b>DONALD</b>  Germany  Libuda et al. (2008)  5 y  Public funding	<p><b>N</b> = 1,170</p> <p><b>Population sampled:</b> General population from Dortmund</p> <p><b>Excluded:</b> age &lt;14 years at the time of last assessment, missing &gt;2 out of six possible dietary records, implausible daily energy intakes, missing data on covariates.</p> <p><b>n</b> = 244 (1316 measurements) Females = 116 Males = 119</p>	<p><b>BMIZ-score and %BF</b></p> <p><b>BW</b> was measured to the nearest 0.1 kg using an electronic scale. <b>Height</b> was measured in a standing position to the nearest 0.1 cm using a digital telescopic stadiometer. Sex- and age-independent BMI SD scores (<b>or BMIZ scores</b>) were</p>	<p><b>g/d</b> <b>Mean ± SD</b> Females: 243 ± 273 Males: 277 ± 296</p> <p><b>Method:</b> 3-d DR</p>	<p>Intake of <b>SSSD+SSFD+SSFJ</b> at baseline and changes in <b>SSSD+SSFD+SSFJ</b> intake over follow-up vs changes in BMIZ-score and %BF over follow-up</p> <p><b>Data collection:</b> every year</p>	<p><b>Model 1:</b> time, age</p> <p><b>Model 2:</b> model 1 + energy from other sources at baseline, change in energy from other sources, weight at birth, years of adolescence, maternal education level, maternal BMI.</p>	<p><b>Females</b> Non-significant <b>positive</b> relationship between baseline intake of SSSD+SSFD+SSFJ, as well as changes in SSSD+SSFD+SSFJ intake over the follow-up, and changes in BMIZ-scores and % BF.</p> <p><b>Males</b> Non-significant <b>positive</b> relationship between baseline intake of SSSD+SSFD+SSFJ and changes in BMIZ-scores. Relationship with % BF was</p>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
		<b>Ethnicity:</b> Caucasian  <b>Age:</b> 9 – 18 y	calculated using the German national reference data <sup>9</sup> .  <b>Triceps and subscapular skinfolds</b> were measured on the right side of the body using a skinfold calliper. The sum of both skinfolds was used for the estimation of <b>%BF</b> according to the equations of Slaughter <sup>10</sup>				<b>negative</b> and non-significant. Non-significant <b>positive</b> relationship between changes in SSSD+SSFD+SSFJ and changes in BMIz-scores and % BF.
<b>3</b>	<b>AGAHLS</b>  The Netherlands  Stoof et al. (2013)  27 y (midpoint of the range)  Mixed funding	<b>N</b> = 409  <b>Population sampled:</b> Children from two secondary schools in Amsterdam and the surrounding area  <b>Excluded:</b> Missing dietary data at baseline, data on weight status and covariates at baseline, data from DXA measurements and BMI at the latest follow-up.  <b>n</b> = 238 Females = 124 Males = 114  <b>Age (mean <math>\pm</math> SD):</b> Females: 12.7 $\pm$ 1 y Males: 12.9 $\pm$ 1.1 y	<b>BMI and %BF</b>  <b>Height and weight</b> measurements were collected at baseline and follow-up by trained research nurses. <b>BMI</b> was defined as body mass (kg) divided by body height squared ( $m^2$ ). If data were available from the two last follow-up, the mean of these two values was calculated. If only data from one of the last follow-up were available, this single value was used in the analysis.	<b>ml/d</b> <b>Mean <math>\pm</math> SD</b> Females: 160 $\pm$ 137 Males: 200 $\pm$ 191  <b>Serving size:</b> 220 ml  <b>Method:</b> DHI	Intake of <b>SSSD+SSFD+SSFJ</b> at baseline and BMI at end of follow-up  <b>Data collection:</b> exposure measured at baseline and ages 14, 15, 16, 21, 27, 29, 32, 36 and 42 y (end of follow-up). Outcome measured at ages of 36 and 42 y.	<b>Model 1:</b> crude  <b>Model 2:</b> BMI at baseline  <b>Model 3:</b> model 2 + developmental age, PAL  <b>Model 4:</b> model 3 + energy intake	<b>Non-significant positive</b> association between baseline intake of SSSD+SSFD+SSFJ and follow-up BMI, for both females and males. <b>Significant positive</b> association between baseline intake of SSSD+SSFD+SSFJ and follow-up %BF in males, but not females ( <b>negative, non-significant</b> ).  <b>BMI</b> <b>Per each 1 serving/day increase <math>\beta</math> coefficients (95% CI), <math>kg/m^2</math></b>  <b>Females</b> <b>Model 1:</b> -0.09 (-1.02, 0.83) <b>Model 2:</b> 0.52 (-0.29, 1.32) <b>Model 3:</b> 0.44 (-0.37, 1.24) <b>Model 4:</b> 0.43 (-0.39, 1.25)

<sup>9</sup> Kromeyer-Hauschild K, Wabitsch M, Kunze D, et al. (2001) Percentiles of body mass index in children and adolescents evaluated from different regional German studies (article in German). Monatsschrift Kinderheilkunde 149, 807–818.

<sup>10</sup> Slaughter MH, Lohman TG, Boileau RA, Horswill CA, Stillman RJ, Van Loan MD & Bembien DA (1988) Skinfold equations for estimation of body fatness in children and youth. Hum Biol 60, 709–723.



RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
							<b>Males</b> Model 1: 0.33 (-0.28, 0.95) Model 2: 0.29 (-0.30, 0.87) Model 3: 0.24 (-0.34, 0.81) Model 4: 0.24 (-0.33, 0.82)  <b>%BF</b> <b>Per each 1 serving/day increase</b> <b>β coefficients (95% CI)</b>  <b>Females</b> Model 1: -1.12 (-2.78, 0.54) Model 2: -0.71 (-2.38, 0.96) Model 3: -0.72 (-2.40, 0.97) Model 4: -0.72 (-2.44, 1.01)  <b>Males</b> Model 1: 1.16 (0.05, 2.26) Model 2: 1.11 (0.01, 2.21) Model 3: 1.10 (-0.02, 2.21) Model 4: 1.14 (0.04, 2.23)
<b>Exposure: SSSD + SSFD + TFJ</b>							
<b>1</b>	<b>HSS-DK</b>  Denmark  Zheng et al. (2015)  1.5 y  Mixed funding	<b>N = 552</b>  <b>Population sampled:</b> Children who had a high predisposition for future overweight based on specific criteria  <b>Excluded:</b> Moving to another municipality after birth, if they were protected from being contacted by researchers, not having a permanent address, living in a childrens' home, moving abroad or having died. Incomplete dietary data and misreporting	<b>BW and BMIZ-score</b> <b>Body weight</b> was measured in underwear to the nearest 0.1 kg using a mechanical weight or a beam-scale type weight. <b>Height</b> was measured barefoot or in stockings to the nearest 0.1 cm using a stature meter. Age- and sex-specific <b>BMIZ-scores</b> were calculated using the Lambda-Mu-Sigma method <sup>11</sup> , and Danish national reference z-scores were	<b>g/d (mean ± SD) †</b> 92 ± 107  1 g ~ 1 ml  <b>Method:</b> 4-d DR	Intake of <b>SSSD+SSFD+TFJ</b> at baseline vs change in BW and BMIZ-score over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> baseline age, BMIZ, sex, intervention allocation, PAL, parents divorced, number of siblings, annual income, maternal education, paternal education, maternal pre-pregnancy overweight, water, milk and diet beverage intake  <b>Model 2:</b> model 1 + energy intake	Every 100 g/d increase in baseline SSSD+SSFD+TFJ intake was <b>significantly (positive)</b> associated with 0.10 kg and 0.06 unit increases in BW and BMI z-score, respectively.  <b>Per 100 g/day increase</b> <b>β coefficients (SE)</b>  <b>BW (kg)</b> Model 1: 0.1 (0.07) <b>P = 0.048</b> Model 2: 0.1 (0.07) <b>P = 0.05</b>  <b>BMIZ-score</b>

<sup>11</sup> Cole TJ & Green PJ (1992) Smoothing reference centile curves: the LMS method and penalized likelihood. Stat Med 11, 1305–1319.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
		energy intake at baseline or follow-up.  <b>n</b> = 352  <b>Sex:</b> 45% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 2 - 6 y	applied to the study population <sup>12</sup>				<b>Model 1:</b> 0.06 (0.03) <b>P</b> < <b>0.04</b> <b>Model 2:</b> 0.06 (0.03) <b>P</b> < <b>0.04</b>  <i>Longitudinal associations of changes in SSB with concurrent changes in body weight and BMIZ-score were NS (data not shown)</i>  <b>Inverse (non-significant) relationship observed for ASB with BMIZ-score and BW.</b>
<b>Exposure: 100% FJ</b>							
<b>1</b>	<b>DONALD</b>  Germany  Libuda et al. (2008)  5 y  Public funding	<b>Same population and exclusion criteria as for SSSD+SSFD+SSFJ</b>	<b>BMIZ-score and %BF</b>  <b>Same ascertainment of outcome as for SSSD+SSFD+SSFJ</b>	<b>g/d</b> <b>Mean ± SD</b>  Females: 180 ± 236 Males: 178 ± 224  <b>Method:</b> 3-d DR	Intake of <b>100% FJ</b> at baseline and changes in <b>100% FJ</b> intake over follow-up vs concurrent changes in BMIZ-score and %BF over follow-up  <b>Data collection:</b> every year	<b>Model 1:</b> time, age  <b>Model 2:</b> model 1 + energy from other sources at baseline, change in energy from other sources, weight at birth, years of adolescence, maternal education level, maternal BMI.  <i>Analysis done using energy derived from 100% FJ (MJ) rather than g/d as exposure is reported</i>	<b>Females</b> Non-significant <b>negative</b> relationship between baseline intake of 100%FJ and changes in BMIZ-scores and % BF. <b>Positive</b> relationship between changes in 100%FJ and changes in BMIZ-scores (significant) and % BF (non-significant). Per each MJ increase intake, BMI z-score increased by 0.096 (SE = NR; p=0.13)  <b>Males</b> Non-significant <b>positive</b> relationship between baseline intake of 100%FJ and changes in BMIZ-scores. Relationship with % BF was <b>negative</b> and non-significant. Non-significant <b>negative</b> relationship between changes in 100%FJ and changes in BMIZ-scores and % BF.

<sup>12</sup> Nysom K, Mølgaard C, Hutchings B, et al. (2001) Body mass index of 0 to 45-y-old Danes: reference values and comparison with published European reference values. Int J Obes Relat Metab Disord 25, 177–184.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
1	HPFS USA Pan et al. (2013) 20 y Public funding	Study population and exclusion criteria as for SSSD+SSFD	<u>BW</u> Ascertainment of outcome as for SSSD+SSFD	<b>servings/d</b> <b>Mean (95% CI)</b> 0.78 (0, 2.43)  <b>Serving size:</b> 177 ml  <b>Method:</b> SFFQ	Change in <b>100%FJ</b> intake vs concurrent change in BW within each 4-y interval over the follow-up  <b>Data collection:</b> every 4 years during follow-up	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + baseline BMI, sleep duration, changes in PAL, alcohol use, TV watching, smoking, other beverages, dietary variables (fruits, vegetables, whole grain, refined grain, potatoes, potato chips, red meat, other dairy products, sweets and desserts, nuts, fried foods and trans-fat)	<b>Significant positive</b> relationship between change in 100%FJ intake and change in BW within each 4-y interval over the follow-up  <b>Per each 1 serving/day increase</b> <b>β coefficients (95% CI), kg</b> <u>Model 1:</u> 0.12 (0.07, 0.16) <u>Model 2:</u> 0.15 (0.10, 0.19)
1	NHS USA Pan et al. (2013) 20 y Public funding	Study population and exclusion criteria as for SSSD+SSFD	<u>BW</u> Ascertainment of outcome as for SSSD+SSFD	<b>servings/d</b> <b>Mean (95% CI)</b> 0.83 (0, 2.29)  <b>Serving size:</b> 177 ml  <b>Method:</b> SFFQ	Change in <b>100%FJ</b> intake vs concurrent change in BW within each 4-y interval over the follow-up  <b>Data collection:</b> every 4 years during follow-up	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + baseline BMI, sleep duration, changes in PAL, alcohol use, TV watching, smoking, other beverages, dietary variables (fruits, vegetables, whole grain, refined grain, potatoes, potato chips, red meat, other dairy products, sweets and desserts, nuts, fried foods and trans-fat)	<b>Significant positive</b> relationship between change in 100%FJ intake and change in BW within each 4-y interval over the follow-up  <b>Per each 1 serving/day increase</b> <b>β coefficients (95% CI), kg</b> <u>Model 1:</u> 0.28 (0.24, 0.32) <u>Model 2:</u> 0.24 (0.20, 0.28)
1	NHS II USA Pan et al. (2013) 16 y Public funding	Study population and exclusion criteria as for SSSD+SSFD	<u>BW</u> Ascertainment of outcome as for SSSD+SSFD	<b>servings/d</b> <b>Mean (95% CI)</b> 0.62 (0, 2.0)  <b>Serving size:</b> 177 ml  <b>Method:</b> SFFQ	Change in <b>100%FJ</b> intake vs concurrent change in BW within each 4-y interval over the follow-up  <b>Data collection:</b> every 4 years during follow-up	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + baseline BMI, sleep duration, changes in PAL, alcohol use, TV watching, smoking, other beverages, dietary variables (fruits, vegetables, whole grain, refined grain, potatoes, potato chips, red meat, other dairy products, sweets and desserts, nuts, fried foods and trans-fat)	<b>Significant positive</b> relationship between change in 100%FJ intake and change in BW within each 4-y interval over the follow-up  <b>Per each 1 serving/day increase</b> <b>β coefficients (95% CI), kg</b> <u>Model 1:</u> 0.22 (0.19, 0.26) <u>Model 2:</u> 0.26 (0.22, 0.30)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
1	WHI USA Auerbach et al. (2018) 3 y Public funding	Same population and exclusion criteria as for SSSD+SSFD+SSFJ	<u>BW</u>  Same ascertainment of outcome as for SSSD+SSFD+SSFJ	Servings/d <sup>+</sup> 0.67 ± 0.63  Serving size: 6oz (177 ml)  Method: SFFQ	Change in <b>100% FJ</b> vs concurrent change in BW (lbs) over the 3-y follow-up  Data collection: baseline and end of follow-up	<b>Model 1:</b> crude  <b>Model 2:</b> age, education, income, ethnicity, current smoking, BMI, HRT, PAL, change in healthy eating index diet quality score  <b>Model 3:</b> model 2 + change in total energy intake	<b>Significant positive</b> association between change in 100% FJ intake and change in BW (lbs) over the 3-y follow-up.  <b>Per each 1 serving/day increase</b> <b>β coefficients (95% CI), lbs</b> Model 1: 0.19 (-0.01, 0.47) Model 2: 0.39 (0.10, 0.69) Model 3: 0.33 (0.04, 0.63)
1	ALSPAC UK Johnson et al. (2007) 4.6 y (mean) Mixed funding	Same population and exclusion criteria as for SSSD+SSFD	<u>BF (kg)</u>  Same ascertainment of outcome as for SSSD+SSFD	g/d Median (IQR) 0 (0, 117)  Serving size: 180 ml  Method: 3-d DR	Intake of <b>100% FJ</b> at 5 and 7 y vs BF at 9 y (end of follow-up)  Data collection: dietary data at 5 and 7 y, BF at 9 y (end of follow-up).	<b>Model 1:</b> sex, height at outcome assessment  <b>Model 2:</b> model 1 + baseline BMI, TV watching, maternal education, paternal class, maternal BMI, paternal BMI, misreporting of energy intake, dietary energy density, %E from fat, fibre density	Significant <b>negative</b> association between intake of 100% FJ at 5 y and BF at 9 y. The association between intake of 100% FJ at 7 y and BF at 9 y was <b>positive</b> (non-significant).  <b>Per each 1 serving/day increase</b> <b>β coefficients (95% CI), kg</b>  <b>5 y</b> Model 1: -0.55 (-1.08, -0.02) Model 2: -0.11 (-0.61, -0.38)  <b>7 y</b> Model 1: -0.22 (-0.66, 0.22) Model 2: 0.25 (-0.08, 0.58)
2	GUTS USA Field et al. (2003)* 3 y	N = 16,882  Population sampled: offspring of participants from NHSII  Excluded: Reported EI <500 or >5000 calories	<u>BMI z-scores</u>  BMI calculated using self-reported <b>height</b> and <b>weight</b> (wt(kg)/ht(m) <sup>2</sup> ) and calculated age- and sex-specific percentiles and <b>z-scores</b> based on the Centers for	Servings/d Mean ± SD Females: 0.8 ± 0.8 Males: 0.9 ± 0.9  Serving size: 237 ml	1-y change in <b>100% FJ</b> vs 1-y change in BMIz-score  Data collection: every year	<b>Model 1:</b> age, age squared, Tanner stage, height change, baseline BMIz score, physical activity and inactivity  <b>Model 2:</b> model 1 + total energy intake	<b>Positive</b> association between 1-y change in 100% FJ intake and 1-y change in BMIz-score, in females ( <b>significant</b> ) and males ( <b>non-significant</b> ) in the most adjusted models including total energy intake.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
	Mixed funding	n = 14,918 Females = 8,203 Males = 6,715  <b>Sex:</b> 55% females  <b>Ethnicity:</b> 94.7% Caucasian, 5.3% other  <b>Age:</b> 9 – 14 y	Disease Control and Prevention and the National Center for Health Statistics growth charts.	<b>Method:</b> SFFQ			<b>Females</b> <b>Per each 1 serving/day increase</b> <b>β coefficients (95% CI):</b> Model 1: -0.000 (-0.002, 0.001) Model 2: 0.003 (0.001, 0.005)  <b>Males</b> <b>Per each 1 serving/day increase</b> <b>β coefficients (95% CI):</b> Model 1: 0.000 (-0.002, 0.002) Model 2: 0.002 (0.000, 0.005)
2	<b>NGHS</b>  USA  Striegel-Moore et al. (2006)  10 y  Unclear funding	<b>N</b> = 2,379  <b>Population sampled:</b> Non-Hispanic Caucasian and African American girls with racially concordant parents from 3 sites  <b>Excluded:</b> not having at least one 3-d DR  <b>Follow-up rate</b> @ 90% <b>n</b> = 2,371  <b>Sex:</b> females <b>Ethnicity:</b> 51% Black, 49% Caucasian <b>Age:</b> 9 – 10 y	<b>BMI</b>  <b>Weight and height</b> were measured annually by research staff.	<b>g/d (mean (SE))</b> NR for pooled cohort  Caucasian v1: 110.46 (4.94) v10: 128.68 (5.42) Black v1: 108.36 (4.86) v10: 119.81 (5.02)  <b>Method:</b> 3-d DR	1-y change in <b>100% FJ</b> intake vs concurrent 1-y change in BMI  <b>Data collection:</b> every year. Each observation refers to two consecutive years.	<b>Model:</b> site, visit, race, total energy intake and consumption of milk, ASSD, fruit juice, coffee/tea, SSFD and SSSD	Non-significant <b>positive</b> association between 1-y change in intake of 100% FJ and 1-y change in BMI  <b>Per each 100 g/d increase β coefficients (SE), kg/m<sup>2</sup></b>  0.005 (0.007), <b>NS</b>
2	<b>MOVE</b>  USA  Carlson et al. (2012)  2 y	<b>Same population and exclusion criteria as for SSSD+SSFD</b>	<b>BMIZ-score and %BF</b>  <b>Same ascertainment of outcome as for SSSD+SSFD</b>	<b>Servings/d Mean ± SD</b> 0.60 ± 0.56  <b>Serving size:</b> 237 ml  <b>Method:</b> SFFQ	Change in <b>100% FJ</b> intake vs concurrent change in BMIZ-score and %BF over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> age, gender, ethnicity, parent education, and height	Non-significant ( <b>negative</b> ) association between change in 100% FJ intake and changes in BMIZ-score and %BF over the follow-up.  <b>Per each serving/d increase β coefficient (95% CI)</b>  <b>BMIZ-score</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
	Public funding						-0.04 (-0.21, 0.13), P = 0.631  <b>%BF</b> -1.06 (-2.70, 0.57), P = 0.202
<b>3</b>	<b>Project Viva</b>  USA  Sonneville et al. (2015)  6.7 y  Mixed funding	<b>N</b> = 2,128  <b>Population sampled:</b> infants from eight urban and suburban obstetric offices in Massachusetts <b>Excluded:</b> no in-person visit during early (3 y) or mid-childhood (7 y).  <b>n</b> = 1163 (model 3 = 1038)  <b>Sex:</b> 49.8% females  <b>Ethnicity:</b> 70.3% Caucasian, 11.7% Black, 3.7% Hispanic, 3.1% Asian and 11.2% other  <b>Age:</b> 1 y	<b>BMI z-scores</b>  <b>Height and weight</b> were measured using a calibrated stadiometer and scale. Age- and sex-specific <b>z-scores</b> calculated using US reference growth data <sup>13</sup> . Research assistants performing all measurements followed standardized techniques <sup>14</sup> and participated in inservice training to ensure measurement validity. Inter- and intra-rater measurement error were within published reference ranges for all measurements <sup>15</sup> .	<b>oz/d</b> <u>G1</u> (ref): 0 <u>G2</u> : 1-7 <u>G3</u> : 8-15 <u>G4</u> : ≥16  1 oz = 29.6 ml  <b>n</b> <u>G1</u> : 262 <u>G2</u> : 619 <u>G3</u> : 235 <u>G4</u> : 47  <b>Method:</b> SFFQ	<b>100% FJ</b> intake at baseline (1 y) vs BMIz-scores at 3 and 7 years.  <b>Data collection:</b> exposure at 1, 3 and 7 years, outcome at 3 and 7 years  <i>Results are reported in this table for the longest follow-up (7 years)</i>	<b>Model 1:</b> crude  <b>Model 2:</b> maternal age, education, prepregnancy BMI, household income, and child age, sex, race/ethnicity, and weight-for-length z-score at baseline  <b>Model 3:</b> model 2 + energy intake at 3 years	<b>Significant positive</b> association between intakes of 100%FJ at 1 y of age and BMIz-scores at 3 and 7 years. Data at 7 years are reported below.  <b>Model 1: β coefficients (95% CI)</b> <u>G1</u> (ref): 0 <u>G2</u> : 0.18 (0.04, 0.33) <u>G3</u> : 0.39 (0.21, 0.57) <u>G4</u> : 0.62 (0.31, 0.92) <b>P for trend &lt;0.0001</b>  <b>Model 2: β coefficients (95% CI)</b> <u>G1</u> (ref): 0 <u>G2</u> : 0.08 (-0.05, 0.20) <u>G3</u> : 0.23 (0.07, 0.39) <u>G4</u> : 0.36 (0.08, 0.64) <b>P for trend = 0.01</b>  <b>Model 3: β coefficients (95% CI)</b> <u>G1</u> (ref): 0 <u>G2</u> : 0.07 (-0.06, 0.21) <u>G3</u> : 0.23 (0.05, 0.40) <u>G4</u> : 0.27 (-0.05, 0.59) <b>P for trend = 0.05</b>

ASSD, artificially sweetened soft drinks; BMI, body mass index; BF, body fatness; BW, body weight; CDC, Centres for Disease Control and Prevention; CI, confidence interval; cm, centimetre; CVD, cardiovascular disease; d, day; DHI, dietary history interview; DR, dietary report; DXA, dual-energy X-ray absorptiometry; EI, energy intake; FJ, fruit juice; kcal, kilocalories; kg, kilogram; kj,

<sup>13</sup> Wardle J, Carnell S, Cooke L. Parental control over feeding and children's fruit and vegetable intake: how are they related? J Am Diet Assoc 2005;105:227-232.

<sup>14</sup> Musher-Eizenman D, Holub S. Comprehensive feeding practices questionnaire: validation of a new measure of parental feeding practices. J Pediatr Psychol 2007;32:960-972.

<sup>15</sup> Mueller W, Martorell R. Reliability and accuracy of measurement. In: Lohman T, Roche A, Martorell R, eds. Anthropometric Standardization Reference Manual. Champaign, IL: Human Kinetics Books; 1988.

kilojoules; IQR, interquartile range; lbs, pounds; MetS, metabolic syndrome; ml, millilitres; MJ, megajoule; n, participants analysed; N, participants included in the cohort; NR, not reported; ns, non-significant; oz, ounce; PAL, physical activity level; PUFA, polyunsaturated fatty acids; SD, standard deviation; SE, standard error; SF, skinfold; SFFQ, semiquantitative food frequency questionnaire; SSB, sugar-sweetened beverages; SSFD, sugar-sweetened fruit drinks; SSFJ, sugar-sweetened fruit juices; SSSD, sugar-sweetened soft drinks; TFJ, total fruit juices; tsp, tea spoon; USA, United States of America; v, visit; WC, waist circumference; wk, week; y, years. \* Data provided by the authors. † Exposure adjusted for total energy intake using the nutrient residuals model. ‡ Adjusted for age and total energy intake. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Continuous variables related to the risk of abdominal obesity: waist circumference, abdominal fat and derived indices

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
<b>Exposure: Total sugars</b>							
<b>1</b>	<b>NGHS</b>  USA  Lee et al. (2015)  6 y  Unclear funding	<p><b>N</b> = 2,379</p> <p><b>Population sampled:</b> Non-Hispanic Caucasian and African American girls with racially concordant parents from 3 sites</p> <p><b>Excluded:</b> Hispanics, pregnancy, pairs of observations where visits were &lt;0.8 or &gt; 1.2 years apart, implausible or invalid nutritional intake; and missing nutrition information, change in BMI, change in WC or other covariates.</p> <p><b>n</b> = 2,021 (5,156 pairs of observations)  <b>n at visits 2-3</b> = 1,597  <b>n at visits 3-4</b> = 1,415  <b>n at visits 4-5</b> = 1,304  <b>n at visits 7-8</b> = 840</p> <p><b>Ethnicity:</b> 51.1% Caucasian and 48.9% Black  <b>Sex:</b> females  <b>Age:</b> 9-10 y</p>	<p><b>WC</b></p> <p>Minimum <b>WC</b> was measured following breath expiration at all visits except baseline (visit 1). The mean of the repeated measures was used for all analysis.</p>	<p><b>Tsp (4g)/d</b>  <b>Mean ± SD</b></p> <p>Visit 2: 25.8 ± 12.9            Visit 3: 27.2 ± 13.0            Visit 4: 26.3 ± 12.5            Visit 7: 28.0 ± 12.6</p> <p><b>Method:</b> 3-d DR</p>	<p>1-y change in <b>total sugar</b> intake vs 1-y change in WC (mm)</p> <p><b>Data collection:</b> every year. Each observation refers to two consecutive years.</p>	<p><b>Model 1:</b> race; initial age, BMI, and puberty stage, parents' income, parents' education, dieting status, initial and change in physical activity, change in height and baseline sugar intake</p> <p><b>Model 2:</b> model 1 + initial and change in grams of fibre, percentage of energy from fat and percentage of energy from other carbohydrates</p> <p><b>Model 3:</b> model 2 + initial and change in total energy intake</p>	<p>Total sugar intake was significantly and <b>positively</b> associated with changes in WC in models 1 and 2. Each teaspoon (4g/d) increase in total sugars intake was associated with a 0.154-mm increase in WC in model 2 (95% CI 0.071, 0.237, p = 0.0003). The association became <b>non-significant</b> (model 3) after adjusting for total energy (0.086 mm, 95% CI = -0.016, 0.187, p = 0.10).</p>



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2	<b>SCES</b>  Australia  Gopinath et al. (2013)  5 y  Mixed funding	<b>N</b> = 2,353  <b>Population sampled:</b> schoolchildren from Sydney  <b>Excluded:</b> NR <b>Follow-up rate:</b> 51.6%  <b>n</b> = 856 <b>Females:</b> 421 <b>Males:</b> 435  <b>Ethnicity:</b> 61.1% Caucasian, 19.5% East Asian, 4% Middle Eastern  <b>Age:</b> 12 y	<b>WC</b>  WC was measured in cm with a measuring tape at the mid-point between the lower rib border and the iliac crest.	<b>Baseline, g/d †</b>  <b>Females, mean (SD)</b> 129.2 ± 55.1  <b>Males (range)</b> <b>T1:</b> ≤120.91 <b>T2:</b> 121.1 – 143.7 <b>T3:</b> ≥143.8  <b>n</b> <b>T1:</b> 141 <b>T2:</b> 142 <b>T3:</b> 152  <b>Method:</b> SFFQ	<b>Total sugars</b> at baseline vs changes in WC over the 5-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> age, ethnicity, parental education, passive smoking, change in energy intake, change in height, screen time and PAL	<b>Non-significant (negative)</b> associations were observed between the intake of total sugars at baseline and changes in WC during the 5-y follow-up after adjustment for confounders in females (analysis with the exposure at baseline as continuous variable). In males (analysis by tertiles of the exposure at baseline), a <b>non-significant (positive)</b> association was reported: <b>Mean, cm (95% CI)</b> <b>T1:</b> 11.73 (10.35, 13.10) <b>T2:</b> 11.45 (9.93, 13.00) <b>T3:</b> 12.08 (10.85, 13.30) <b>P for trend</b> = 0.49
<b>Exposure: free and/or added sugars</b>							
2	<b>Mr and Ms OS</b>  China  Liu et al. (2018)  4 y  Public funding	<b>N</b> = 4,000  <b>Population sampled:</b> General population  <b>Excluded:</b> Unable to walk independently or with bilateral hip replacements, diabetes at baseline.  <b>Follow-up rate:</b> 75%  <b>n</b> = 3,421 Females = 1,714 Males = 1,707  <b>Ethnicity:</b> Asian  <b>Age:</b> ≥65 y	<b>Abdominal fat (kg)</b>  Body fat was measured by DXA. In measuring the trunk fat, a line of delineation was drawn between the head of the humerus and the glenoid fossa of the scapula to separate the upper limb from the trunk and another line passed through the femoral necks and just below the ischium to separate the pelvis from the leg. The android region is the area between the ribs and the pelvis, and this region is totally enclosed by the trunk region. Abdominal fat was estimated by adding fat in the android and trunk regions.	<b>%E Mean ± SD</b>  <b>Free sugars</b> Females: 4.1 ± 3.8 Males: 4.6 ± 3.5  <b>Added sugars</b> Females: 3.0 ± 3.2 Males: 3.6 ± 3.0  <b>Method:</b> SFFQ	<b>Free and added sugars</b> at baseline vs changes in abdominal fat over the 4-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> crude  <b>Model 2:</b> age, weight, history of CVD, monthly income, physical activity, education, smoking, and dietary intakes of whole grains, fruits and vegetables, red and processed meat, alcohol, green and Chinese tea, and caffeine	<b>Significant positive</b> associations between intakes of free and added sugars at baseline and changes in abdominal fat over follow-up in <b>males</b> . Non-significant positive associations in females.  <b>Per each 1%E increase β coefficients (SE), kg</b>  <b>Free sugars, males</b> <b>Model 1:</b> 0.022 (0.01) <b>Model 2:</b> 0.027 (0.01)  <b>Added sugars, males</b> <b>Model 1:</b> 0.023 (0.012) <b>Model 2:</b> 0.029 (0.012)  <b>Free sugars, females</b> <b>Model 1:</b> 0.013 (0.009) <b>Model 2:</b> 0.013 (0.009)

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							<b>Added sugars, females</b> <b>Model 1:</b> 0.017 (0.01) <b>Model 2:</b> 0.017 (0.01)
1	<b>NGHS</b>  USA  Lee et al. (2015)  6 y  Unclear funding	<b>Study population and exclusion criteria as for total sugars</b>	<b>WC</b>  <b>Ascertainment of outcome as for total sugars</b>	<b>tsp/d (mean <math>\pm</math> SD)</b> Baseline: 21.0 $\pm$ 11.8 Follow-up 1: 22.3 $\pm$ 12.0 Follow-up 2: 22.1 $\pm$ 11.5 Follow-up 3: 22.6 $\pm$ 11.7  <b>Serving size:</b> 1 tsp = 4g  <b>Method:</b> 3-d DR	1-y change in <b>added sugars</b> intake vs 1-y change in WC (mm)  <b>Data collection:</b> every year. Each observation refers to two consecutive years.	<b>Model 1:</b> race, initial age, initial BMI, initial puberty stage, parents' income, parents' education, dieting status, initial and change in physical activity, change in height and baseline sugars  <b>Model 2:</b> model 1 + initial and change in grams of fibre, percentage of energy from fat and percentage of energy from other carbohydrates  <b>Model 3:</b> model 2 + initial and change in total energy intake	A significant <b>positive</b> association between change in of added sugars intake and change in WC over 1 y.  <b>Per each 1 tsp/d (4 g/d) increase</b> <b>B coefficients (95% CI), mm</b> <b>Model 1:</b> 0.130 (0.054, 0.205) <b>Model 2:</b> 0.179 (0.093, 0.265) <b>Model 3:</b> 0.107 (0.002, 0.212)
1	<b>QUALITY</b>  USA  Wang et al. (2014)  2 y  Public funding	<b>N = 630</b>  <b>Population sampled:</b> General population from Quebec with at least one biological parent that had obesity and/or abdominal obesity  <b>Excluded:</b> Diabetes, following a very restricted diet (< 2510 kJ/d), regular medication use, and serious psychological ailments.  <b>Follow-up rate:</b> 97% <b>n = 472</b> <b>Sex:</b> 44.5 % females <b>Ethnicity:</b> Caucasian <b>Age (range):</b> 8 – 10 y	<b>WC</b>  <b>WC</b> was measured using a standard measurement tape following a standard protocol.	<b>g/d from liquid sources Mean <math>\pm</math> SD</b> 11.4 $\pm$ 12.5  <b>g/d from solid sources Mean <math>\pm</math> SD</b> 40.4 $\pm$ 22.2  <b>Method:</b> Three 24-h DR	<b>Added sugars from liquid and solid sources</b> at baseline vs changes in WC over the 2-y follow-up  <b>Data collection:</b> exposure at baseline, outcome at baseline and end of follow-up	<b>Model:</b> baseline WC, age, sex, tanner stage, energy intake, fat mass index and physical activity.	<b>Non-significant negative</b> association between the intake of added sugars from solid sources and changes in WC over follow-up. Association was also non-significant but <b>positive</b> for added sugars from liquids.  <b>Per each 10 g/d increase</b> <b>BMI, <math>\beta</math> coefficients (95% CI), cm</b> <b>Liquid sources</b> 0.159 (-0.214, 0.531) <b>Solid sources</b> -0.076 (-0.330, 0.179)
<b>Exposure: sucrose</b>							

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
2	<b>EPIC-Norfolk</b>  UK  Kuhnle et al. (2015)  3 y  Public funding	<b>N</b> = 25,639  <b>Population sampled:</b> Norfolk's inhabitants  <b>Excluded:</b> Missing co-variables (i.e. sex, dietary data, second health check anthropometry), urinary sucrose analysis failed or outside the calibration range  <b>n</b> = 1,734 Females = 937 Male = 797  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 39 – 79 y	<b>WC</b>  <b>WC</b> was measured at follow-up by trained research nurses using a standardised protocol.	<b>g/d † Geometric mean (SD)</b> Females: 45.0 (20.8) Males: 58.3 (29.1)  <b>g/MJ/d (range)</b> Females: 0.1 - 16.5 Males: 0.3 - 19.1  <b>% contribution to total sugars Geometric mean (SD)</b> Females: 43 (10) Males: 46 (12)  <b>Methods:</b> 24-h recall + 6-d DR = 7DD Urinary sucrose (spot urine)	<b>Sucrose</b> intake (7DD) and sucrose in urine at baseline vs WC at the end of follow-up  <b>Data collection:</b> baseline for the exposure, baseline and end of follow-up for the outcome	<b>Model 1:</b> age, height  <b>Model 2:</b> model 1 + physical activity	<b>Significant negative</b> associations between baseline sucrose intake and follow-up WC for males and females.  <b>7DD</b> <b>Per each 1 log(g/MJ/day) increase</b> <b>β coefficients (95% CI), cm</b>  <b>Females</b> <b>Model 1:</b> -4.20 (-5.75, -2.64) <b>Model 2:</b> -4.06 (-5.61, -2.50)  <b>Males</b> <b>Model 1:</b> -3.35 (-4.78, -1.93) <b>Model 2:</b> -3.27 (-4.70, -1.85)  <i>Associations between urinary sucrose and WC were in the opposite direction (positive, significant for females).</i>
<b>Exposure: fructose</b>							
2	<b>SCES</b>  Australia  Gopinath et al. (2013)  5 y  Mixed funding	<b>Same population and exclusion criteria as for total sugars</b>	<b>WC</b>  <b>Same ascertainment of outcome as for total sugars</b>	<b>Baseline, g/d †</b>  <b>Females, NR</b>  <b>Males (range)</b> T1: ≤26.1 T2: 26.2 – 34.6 T3: ≥34.7  <b>n</b> T1: 161 T2: 141 T3: 133  <b>Method:</b> SFFQ	<b>Females:</b> changes in fructose intake vs changes in WC over the 5-y follow-up  <b>Males:</b> Intake of fructose at baseline vs changes in WC over the 5-y follow-up  <b>Data collection:</b>	<b>Model:</b> age, ethnicity, parental education, passive smoking, change in energy intake, change in height, screen time and PAL	In females, a <b>non-significant</b> (p=0.08) <b>increase</b> in WC of 1.18 cm (SE = 0.66) was reported for each SD increase (14.2 g/d) in fructose intake over the 5 years of follow-up. In males (analysis by tertiles of fructose intake at baseline vs changes in WC over 5 years), a <b>non-significant (positive)</b> association was reported:  <b>Mean (95% CI), cm</b> T1: 11.60 (10.15, 13.04) T2: 11.57 (10.55, 12.59) T3: 12.16 (10.25, 14.07)

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					baseline and end of follow-up		<b>P for trend = 0.32</b>
<b>3</b>	<b>TLGS</b>  Iran  Bahadoran et al. (2017)  6.7 y (mean)  Public funding	<b>N</b> = 15,005  <b>Population sampled:</b> general population from one district of Tehran  <b>Excluded:</b> Unusual energy intake (<800 kcal/day or >4200 kcal/day, respectively), or were on specific diets for hypertension, diabetes or dyslipidemia; those with a history of CVD at baseline.  <b>n</b> = 2,369  <b>Follow-up rate:</b> 99.5%  <b>Sex:</b> 56.5% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> ≥ 19 y	<b>WC</b>  <b>WC</b> was measured to the nearest 0.1 cm, midway between the lower border of the ribs and the iliac crest at the widest portion, over light clothing, using a soft measuring tape, without any pressure to the body.	<b>%E</b> <b>Mean ± SD</b> 6.4 ± 3.7  <b>Method:</b> SFFQ	Intake of <b>fructose at baseline</b> vs changes in WC (cm) over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> age	Per each 1 %E increase in fructose intake at baseline, the mean increase in WC over the mean follow-up of 6.7 years was 0.387cm (95% CI = 0.252, 0.522). This <b>positive association</b> between fructose intake and changes in WC was <b>statistically significant</b> .
<b>Exposure: SSSD</b>							
<b>2</b>	<b>MTC</b>  Mexico  Stern et al. (2017)*  2 y  Unclear funding	<b>N</b> = 27,992  <b>Population sampled:</b> female teachers  <b>Excluded:</b> Diabetes, cancer, heart disease, ≥65 years, inadequate dietary information (energy intake <500 or >3500 kcal/day, response to ≤70 items in the dietary questionnaire, or missing cereal section), women with missing information on soda consumption in either 2006 or 2008. Women for	<b>WC</b>  Participants self-reported <b>WC</b> (cm) with a plastic measuring tape and instructions. Reproducibility and validity of self-reported anthropometry was evaluated in a subset of 3,413 participants. Standardized technician measurements were well correlated with self-reported waist circumference (r = 0.78). Changes in WC were calculated by subtracting self-	<b>Servings/d (mean ± SD)</b> 0.4 ± 0.5  <b>Change in servings/week from baseline (actual change; mean ± SD)</b> <b>G1:</b> < -1 (-3.7 ± 2.0) <b>G2 (ref):</b> -1 to 1 (-0.1 ± 0.4) <b>G3:</b> >1 (2.8 ± 1.1)	Change in <b>SSSD</b> intake vs changes in WC over the 2-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> baseline soda consumption (sugar and sugar-free), age, state (area), PAL, smoking, alcohol, changes in smoking and alcohol consumption, HRT, menopausal status, oral contraceptives, red meat, dairy, yogurt, fruit, nuts, vegetables, white bread, flour tortillas, corn tortillas, orange and grapefruit juice, homemade sweetened beverages	A significant positive relationship was observed between changes in SSSD intake and changes in WC over the 2-y follow-up. Each serving/day increase in SSSD intake was associated with an increase of 0.9 cm (95% CI: 0.5, 1.4) in WC.  <b>β coefficients (95% CI) cm</b> <b>G1:</b> -0.5 (-0.9, -0.1) <b>G2 (ref):</b> 1 <b>G3:</b> 0.3 (0.1, 0.6)

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		whom BMI could not be calculated because of missing height or weight  <b>n</b> = 9,294  <b>Sex:</b> females  <b>Ethnicity:</b> Hispanic  <b>Age:</b> ≥ 25 y	reported measures in 2008 from those in 2006.	<b>n</b> <b>G1:</b> 2,538 <b>G2:</b> 5,350 <b>G3:</b> 1,406  <b>Serving size:</b> 355 ml  <b>Method:</b> SFFQ			<b>Significant inverse relationship observed for ASSD</b>
3	<b>DCH</b>  Denmark  Olsen et al. (2016)  5 y  Mixed funding	<b>N</b> = 57,053  <b>Population sampled:</b> Inhabitants from Copenhagen and Aarhus counties  <b>Excluded:</b> If aged >60 y at baseline and aged >65 y at follow-up, history of cancer or developed cancer, CVD, or diabetes during the study period, unstable smoking habits between baseline and follow-up, and had a mean gain in BW >5 kg/y.  <b>n</b> = 2,126  <b>Sex:</b> 49.4% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 50 – 64 y	<b>WC and WC<sub>BMI</sub></b>  <b>WC</b> was measured horizontally midway between the lower rib margin and the iliac crest to the nearest 1 cm at baseline, whereas follow-up WC was provided as a self-reported measure after the receipt of instructions at the level of the umbilicus. <b>WC<sub>BMI</sub></b> was defined as residuals of WC regressed on BMI (sex- and study specific regressions; separately for baseline and follow-up values).	<b>ml/d median (95% CI)</b> 10.5 (0.3 - 200.3)  <b>Method:</b> SFFQ	Intake of <b>SSSD</b> at baseline vs annual changes in WC and <b>WC<sub>BMI</sub></b> over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> baseline WC/ <b>WC<sub>BMI</sub></b> , height, sex, age, smoking status, alcohol consumption, PAL, education, menopausal status  <b>Model 2:</b> model 1 + energy intake	Non-significant <b>positive</b> association between the baseline intake of SSSD and annual changes in WC over follow-up. The association was <b>negative</b> (non-significant) for annual changes in <b>WC<sub>BMI</sub></b> .  <b>WC</b> <b>Per 200 ml/d increase β coefficients (95% CI) cm/y</b> <b>Model 1:</b> 0.03 (-0.10, 0.15) <b>Model 2:</b> 0.03 (-0.09, 0.16)  <b>WC<sub>BMI</sub></b> <b>Per 200 ml/d increase β coefficients (95% CI) cm/y</b> <b>Model 1:</b> -0.02 (-0.13, 0.08) <b>Model 2:</b> -0.02 (-0.13, 0.08)
3	<b>Inter99</b>  Denmark  Olsen et al. (2016)  2 y	<b>N</b> = 13,016  <b>Population sampled:</b> Inhabitants from Copenhagen county  <b>Excluded:</b> Prevalent cancer, CVD, or self-reported diabetes at baseline	<b>WC and WC<sub>BMI</sub></b>  Baseline and follow-up <b>WC</b> was measured horizontally midway between the lower rib margin and the iliac crest to the nearest 1 cm. <b>WC<sub>BMI</sub></b> was	<b>ml/d median (95% CI)</b> 16.4 (0, 500)  <b>Method:</b> SFFQ	Intake of <b>SSSD</b> at baseline vs annual changes in WC and <b>WC<sub>BMI</sub></b> over the follow-up	<b>Model 1:</b> baseline WC/ <b>WC<sub>BMI</sub></b> , height, sex, age, smoking status, alcohol consumption, PAL, education, menopausal status	Non-significant <b>positive</b> association between the baseline intake of SSSD and annual changes in WC and <b>WC<sub>BMI</sub></b> over follow-up.  <b>WC</b> <b>Per 200 ml/d increase</b>

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	Mixed funding	or had incident cancer, CVD or self-reported diabetes during follow-up.  <b>n</b> = 1,254  <b>Sex:</b> 49.3% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 30 – 60 y	defined as residuals of WC regressed on BMI (sex- and study specific regressions; separately for baseline and follow-up values).		<b>Data collection:</b> baseline and end of follow-up	<b>Model 2:</b> model 1 + energy intake	<b>β coefficients (95% CI) cm/y</b> <u>Model 1:</u> -0.02 (-0.23, 0.19) <u>Model 2:</u> 0.02 (-0.20, 0.24)  <b>WC<sub>BMI</sub></b> <b>Per each 200 ml/d increase</b> <b>β coefficients (95% CI) cm/y</b> <u>Model 1:</u> 0.05 (-0.09, 0.2) <u>Model 2:</u> 0.09 (-0.06, 0.24)
3	<b>EPIC-DiOGenes</b>  IT, UK, NL, DE, DK  Romaguera et al. (2011)  5.5 y (median)  Public funding	<b>N</b> = 146,543  <b>Population sampled:</b> General population from 5 countries (8 sites)  <b>Excluded:</b> No blood samples collected, age at baseline >60 years or age at follow-up >65 years, pregnant women, missing information on smoking or changing smoking status between baseline and follow-up, missing information on diet or anthropometrics, participants in the lowest and highest 1% of the EPIC cohort distribution of the ratio of reported total energy intake: energy requirement, individuals with prevalent chronic diseases (cancer, diabetes and/or cardiovascular disease) at baseline, incident chronic diseases during follow-up and those with unrealistic anthropometric measurements.  <b>Follow-up rate:</b> 69.8%  <b>n</b> = 48,631 <b>Females:</b> 28,937 <b>Males:</b> 19,694	<b>WC<sub>BMI</sub></b>  <b>WC<sub>BMI</sub></b> was defined as the residual values from the gender- and centre-specific regression equations of WC on BMI. <b>WC</b> (cm) was measured either at the midway between the lowest rib and the iliac crest (the Netherlands, and Potsdam-Germany) or at the narrowest torso circumference (the other centres). At follow-up, participants in UK and the Netherlands (Doetinchem) were measured by trained technicians using the same protocols as at baseline, whereas other centres provided self-reported data. For the latter, guidance was provided to measure WC as at baseline, except for Denmark in which participants were guided to measure their WC at the umbilicus.  Validity of the self-reported WC was assessed in 408 Danish adults. A high	<b>g/d mean ± SD (range)</b> Females: 863.22 ± 525 (154.84–1122.60)  Males: 959.76 ± 501.82 (139.59–1138.79)  <b>Method:</b> SFFQ	Intake of <b>SSSD</b> at baseline vs annual changes in WC <sub>BMI</sub> over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> centre-specific analysis adjusted for total energy intake, age, baseline weight, height, baseline outcome, smoking, alcohol intake, PAL, education, follow-up duration, menopausal status, HRT	<b>Significant positive</b> association between intake of SSSD at baseline and annual changes in WC <sub>BMI</sub> for both males and females over the follow-up.  <b>Per each 100 kcal/day increase</b> <b>β coefficients (95% CI) cm</b> <u>Females:</u> 0.05 (0.02, 0.09) <u>Males:</u> 0.02 (0.00, 0.04)  <i>Soft drinks combines both sugar- and artificially sweetened soft drinks. As results are given per each 100kcal/day increase in intake, it is assumed that the contribution to energy comes predominantly from SSBs.</i>

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		<b>Ethnicity:</b> Caucasian <b>Age:</b> 20-60 y	correlation between self-reported and technician measured WC was found.				
<b>Exposure: SSSD+SSFD</b>							
<b>1</b>	<b>ALSPAC</b>  UK  Bigornia et al. (2015)  3 y (mean)  Mixed funding	<b>N</b> = 14,541  <b>Population sampled:</b> General population living within a defined part of the country  <b>Excluded:</b> missing anthropometric, DXA, dietary and/or physical activity information  <b>n</b> = 2,455 (model 4 = 1,059)  <b>Sex:</b> 53.0% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 10 y	<b>WC</b>  <b>WC</b> was measured to the nearest millimetre at the midpoint between the lowest rib and the top of the iliac crest	<b>Servings/d Median (IQR)</b> Females: 0.3 (1.0) Males: 0.4 (1.4)  <b>Change in servings/day from baseline: Mean (SD)</b> 0.12 (1.36)  <b>Serving size:</b> 180 ml  <b>Method:</b> 3-d DR	Change in <b>SSSD+SSFD</b> intake vs change in WC over the 3-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> change in intake from baseline, baseline SBB intake, sex, baseline age, height and adiposity (not defined)  <b>Model 2:</b> model 1 + PAL at 13y, pubertal stage at 13y, maternal overweight/obesity, maternal education, dieting at 13y, change from baseline in fruit juice, fruit, vegetable and fat intake  <b>Model 3:</b> model 2 + dietary reporting errors at 13y  <b>Model 4:</b> model 2 among plausible dietary reporters at 13y	<b>Significant positive</b> associations between change in intake of SSSD+SSFD and change in WC over the 3-y follow-up after accounting for dietary misreporting. Association was attenuated by 22% when adjusting for total energy in sensitivity analyses and was independent from baseline consumption of SSSD+SSFD. The association was weakened, but remained statistically significant after accounting for BMI ( $\beta = 0.24$ , $P = 0.02$ ) and BF ( $\beta = 0.27$ , $P = 0.01$ ).  <b>Per each 1 serving/d increase <math>\beta</math> coefficients (SE), cm</b> <u>Model 1:</u> 0.12 (0.10) $P = 0.207$ <u>Model 2:</u> 0.13 (0.10) $P = 0.188$ <u>Model 3:</u> 0.22 (0.10) $P = 0.025$ <u>Model 4:</u> 0.55 (0.14) $P < 0.001$
<b>Exposure: SSSD + SSFD + SSFJ</b>							
<b>2</b>	<b>WAPCS</b>  Australia  Ambrosini et al. (2013)  3 y	<b>N</b> = 2,868  <b>Population sampled:</b> offspring from mothers from the Raine study  <b>Excluded:</b> Subjects who reported not fasting before venepuncture.  <b>n</b> = 1,360 Females = 656	<b>WC</b>  <b>WC</b> was measured at the level of the umbilicus to the nearest 0.1 cm, and the average of 2 measurements was used.	<b>g/d mean <math>\pm</math> SD (range)</b> T1 (ref): 48 $\pm$ 39 (0 – 130) T2: 223 $\pm$ 59 (130 – 329) T3: 665 $\pm$ 351 (331 – 2,876)	Changes in <b>SSSD+SSFD+SSFJ</b> intake vs percent of change in WC over the 3-y follow-up  <b>Data collection:</b>	<b>Model 1:</b> age, pubertal stage, physical fitness, dietary misreporting, maternal education, family income  <b>Model 2:</b> model 1 + BMI	<b>Positive</b> associations between change in SSSD+SSFD+SSFJ intake and change in WC in males ( <b>significant</b> ) and females ( <b>non-significant</b> ) over the 3-y follow-up. While this association was also significant for females in model 2, adjustment for western dietary patterns attenuated this relation and became non-significant.



RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
	Unclear funding	Males = 704  <b>Sex:</b> 48.2% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 14 y		<i>n of those who changed tertiles between 14 and 17 y NR</i>  <b>Method:</b> SFFQ	baseline and end of follow-up	<b>Model 3:</b> model 2 + healthy and Western diet pattern scores	<b>Per each tertile of intake increase Δ% (95% CI) vs T1</b>  <b>Females</b> <b>Model 1:</b> T2: 1.2 (-0.3, 2.7) T3: 4.2 (2.5, 5.9) <b>P for trend &lt;0.001</b>  <b>Model 2:</b> T2: 0.9 (0.02, 1.8) T3: 1.2 (0.2, 2.2) <b>P for trend = 0.011</b>  <b>Model 3:</b> T2: 0.8 (-0.1, 1.7) T3: 0.9 (-0.2, 2.0) <b>P for trend = 0.07</b>  <b>Males</b> <b>Model 1:</b> T2: 2.1 (0.5, 3.6) T3: 2.3 (0.7, 4.0) <b>P for trend = 0.007</b>  <b>Model 2:</b> T2: 1.3 (0.4, 2.3) T3: 1.2 (0.3, 2.2) <b>P for trend = 0.019</b>  <b>Model 3:</b> T2: 1.3 (0.3, 2.2) T3: 1.4 (0.2, 2.3) <b>P for trend = 0.025</b>
<b>3</b>	<b>AGAHLS</b>  The Netherlands	<b>N</b> = 409  <b>Population sampled:</b> Children from two secondary schools in	<b>%Trunk fat</b>  In 2000 and 2006, total body <b>FM</b> was measured using DXA.	<b>ml/d</b> <b>Mean ± SD</b> Females: 160 ± 137	Intake of <b>SSSD+SSFD+SSFJ</b> at baseline vs % trunk fat	<b>Model 1:</b> crude  <b>Model 2:</b> BMI at baseline	<b>Significant positive</b> association between the baseline intake of SSSD+SSFD+SSFJ and follow-up



RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
	Stoof et al. (2013)  27 y (midpoint of the range)  Mixed funding	Amsterdam and the surrounding area  <b>Excluded:</b> Missing dietary data at baseline, data on weight status and covariates at baseline, data from DXA measurements and BMI at the latest follow-up.  <b>n</b> = 238 Females = 124 Males = 114  <b>Age (mean <math>\pm</math> SD):</b> Females: 12.7 $\pm$ 1 y Males: 12.9 $\pm$ 1.1 y	If data were available from the two last follow-up, the mean of these two values was calculated. If only data from one of the last follow-up were available, this single value was used in the analysis.	Males: 200 $\pm$ 191  <b>Serving size:</b> 220 ml  <b>Method:</b> DHI	at end of follow-up  <b>Data collection:</b> exposure measured at baseline and the ages of 14, 15, 16, 21, 27, 29, 32, 36 and 42 y (end of follow-up). Outcome measured at ages of 36 and 42 y.	<b>Model 3:</b> model 2 + developmental age, PAL  <b>Model 4:</b> model 3 + energy intake	%trunk fat in males but not in females (n.s <b>negative</b> ).  <b>Females</b> <b>Per each 1 serving/day increase</b> <b><math>\beta</math> coefficients (95% CI) %</b> <u>Model 1:</u> -1.14 (-3.20, 0.92) <u>Model 2:</u> -0.74 (-2.83, 1.36) <u>Model 3:</u> -0.77 (-2.88, 1.35) <u>Model 4:</u> -0.85 (-3.02, 1.31)  <b>Males</b> <b>Per each 1 serving/day increase</b> <b><math>\beta</math> coefficients (95% CI) %</b> <u>Model 1:</u> 1.66 (0.17, 3.16) <u>Model 2:</u> 1.61 (0.13, 3.10) <u>Model 3:</u> 1.57 (0.07, 3.08) <u>Model 4:</u> 1.62 (0.14, 3.10)
<b>Exposure: TFJ</b>							
<b>3</b>	<b>EPIC-DiOGenes</b>  IT, UK, NL, DE, DK  Romaguera et al. (2011)  5.5 y (median)  Public funding	<b>Same population and exclusion criteria as for SSSD</b>	<b>WC<sub>BMI</sub></b>  <b>Same ascertainment of outcome as for SSSD</b>	<b>g/d mean <math>\pm</math> SD (range)</b> Females: 76.50 $\pm$ 128.63 (35.24–199.77)  Males: 63.76 $\pm$ 117.91 (31.19–189.97)  <b>Method:</b> SFFQ	Intake of <b>TFJ</b> at baseline vs annual changes in WC <sub>BMI</sub> over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> centre-specific analysis adjusted for total energy intake, age, baseline weight, height, baseline outcome, smoking, alcohol intake, PAL, education, follow-up duration, menopausal status, HRT	Non-significant <b>negative</b> association between intake of TFJ at baseline and annual changes in WC <sub>BMI</sub> for both males and females over the follow-up.  <b>Per each 100 kcal/day increase</b> <b><math>\beta</math> coefficients (95% CI) cm</b> <u>Females:</u> -0.02 (-0.05, 0.01) <u>Males:</u> -0.01 (-0.02, 0.01)

ASSD, artificially sweetened soft drinks; BMI, body mass index; BF, body fatness; BW, body weight; CDC, Centres for Disease Control and Prevention; CI, confidence interval; cm, centimetre; CVD, cardiovascular disease; d, day; DHI, dietary history interview; DR, dietary report; DXA, dual-energy X-ray absorptiometry; EI, energy intake; FJ, fruit juice; kcal, kilocalories; kg, kilogram; kj, kilojoules; IQR, interquartile range; lbs, pounds; MetS, metabolic syndrome; ml, millilitres; MJ, megajoule; n, participants analysed; N, participants included in the cohort; NR, not reported; ns, non-significant; oz, ounce; PAL, physical activity level; PUFA, polyunsaturated fatty acids; SD, standard deviation; SE, standard error; SF, skinfold; SFFQ, semiquantitative food frequency questionnaire;

SSB, sugar-sweetened beverages; SSFD, sugar-sweetened fruit drinks; SSFJ, sugar-sweetened fruit juices; SSSD, sugar-sweetened soft drinks; TFJ, total fruit juices; tsp, tea spoon; USA, United States of America; v, visit; WC, waist circumference;  $WC_{BMI}$  = WC regressed on BMI; wk, week; y, years. \* Data provided by the authors. † Exposure adjusted for total energy intake using the nutrient residuals model. *Unless otherwise noted, all of the above are prospective cohort studies.*

## Incidence of overweight and/or obesity and incidence of abdominal obesity

### Incidence of overweight and/or obesity

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: SSSD</b>							
<b>1</b>	<b>BWHS</b>  USA  Boggs et al. (2013)  14 y  Public funding	<b>N</b> = 59,001  <b>Population sampled:</b> African American women from all regions of USA  <b>Excluded:</b> pregnant at baseline; history of cancer (except nonmelanoma skin cancer), CVD or gastric surgery; > 10 items blank on the baseline FFQ; implausible energy intake values (<400 or >3800 kcal); missing weight on all follow-up questionnaires; BMI <18.5 or $\geq 30$ kg/m <sup>2</sup> at baseline.  <b>n</b> = 19,479 <b>Sex:</b> females <b>Ethnicity:</b> African American <b>Age:</b> 21-39 y	<b>Incidence of obesity</b> Height and weight reported by participants.  Validation study indicated excellent correlation between self-reported and measured values for height and weight (r=0.93 and r=0.97 respectively).  Obesity defined as BMI $\geq 30$ kg/m <sup>2</sup> .	<b>Servings/time (range)</b> <b>C1</b> (ref): <1/mo <b>C2:</b> 1-7/mo <b>C3:</b> 2-6/wk <b>C4:</b> 1/d <b>C5:</b> $\geq 2$ /d  Serving size = 12 oz (355ml)  <b>Person-years</b> <b>C1</b> (ref): 49,640 <b>C2:</b> 69,282 <b>C3:</b> 46,339 <b>C4:</b> 15,104 <b>C5:</b> 12,444  <b>Exposure assessment:</b> SFFQ	<b>C1</b> (ref): 1,616 <b>C2:</b> 2,436 <b>C3:</b> 1,736 <b>C4:</b> 614 <b>C5:</b> 550	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + baseline BMI, vigorous physical activity, walking for exercise, education, geographic region, <i>smoking status</i> , alcohol intake, parity, prudent and Western dietary patterns	<b>Model 1; HR (95%CI)</b> <b>C1</b> (ref): 1 <b>C2:</b> 1.08 (1.02, 1.25) <b>C3:</b> 1.15 (1.07, 1.23) <b>C4:</b> 1.25 (1.14, 1.38) <b>C5:</b> 1.36 (1.24, 1.50) <b>P per trend</b> = <0.001  <b>Model 2; HR (95%CI)</b> <b>C1</b> (ref): 1 <b>C2:</b> 1.05 (0.98, 1.12) <b>C3:</b> 1.03 (0.95, 1.11) <b>C4:</b> 1.08 (0.98, 1.20) <b>C5:</b> 1.12 (1.00, 1.25) <b>P per trend</b> =0.07
<b>Exposure: SSSD + SSFD</b>							
<b>2</b>	<b>DDHP</b>  USA	<b>N</b> = 1,021  <b>Population sampled:</b> low-income	<b>Incidence of overweight or obesity</b> Weighted on a calibrated digital scale and heights measured from a wall-	<b>oz/d (mean <math>\pm</math> SE)</b> Baseline 19.2 $\pm$ 1 Follow-up	<b>Incidence of overweight:</b> 75 (26.3%)	<b>Model 1:</b> crude  <b>Model 2:</b> model 1 + BMI, age, gender, caregiver's education and	<b>Incidence of overweight or obesity</b>  <b>OR (95%CI) per oz/day of beverage intake at baseline</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Lim et al. (2009)  2 y  Mixed funding	African American children from Detroit  <b>Excluded:</b> energy intake <750kcal or >6500kcal/d, protein intake <19 g/d, calcium intake >4,000 mg/d, vitamin C intake 20% higher than the other children.  <u>For incidence of overweight:</u> excluded those being overweight or obese at baseline.  <b>n = 275</b>  <u>For incidence of obesity:</u> excluded those being obese at baseline.  <b>n = 325</b>  <b>Sex:</b> 51.6% females <b>Ethnicity:</b> black <b>Age:</b> 3-5 y	mounted tape measure, following standard protocol from the NHANES. BMI converted to BMI Z-score using BMI percentiles for each child obtained from Centers for Disease Control and Prevention 2000 growth charts. Children classified as not overweight (BMI <85 <sup>th</sup> percentile), overweight (BMI ≥85 <sup>th</sup> percentile and <95 <sup>th</sup> percentile) and obese (BMI ≥95 <sup>th</sup> percentile).	21.6±1.1  1 oz @ 29.6 mL  <b>Exposure assessment:</b> SFFQ	<b>Incidence of obesity:</b>  51 (13.4%)	income, and child's baseline total energy intake  <b>Model 3:</b> model 2 + caregiver's BMI	<u>Model 1:</u> 1.02 (1.00, 1.04) <u>Model 2:</u> 1.04 (1.01, 1.06) <u>Model 3:</u> 1.04 (1.01, 1.07)  <b>Incidence of obesity</b> Positive and NS (data not shown)
2	PHI  USA  Ludwig et al. (2001)  19 mo	<b>N = 780</b>  <b>Population sampled:</b> Children from four communities in the Boston metropolitan area	<b>Incidence of obesity</b> Height measured to the nearest 0.1cm using a Shorr stadiometer and weight was measured to the nearest 0.1 kg on a portable electronic scale.  Obesity was defined with a composite indicator, based	<b>Servings/d (mean ± SD)</b>  Baseline: 1.22±1.10 Follow-up: 1.44±1.09  Serving size: 12 oz (355 mL)	37 (9.3%)	<b>Model 1:</b> age, sex, ethnicity, BMI and triceps-skinfold thickness  <b>Model 2:</b> model 1 + baseline values and changes from baseline to follow-up of the following variables: %E from fat, energy-adjusted fruit-juice intake, physical activity, television viewing	<b>OR (95%CI) per each serving at baseline</b> <u>Model 1:</u> 1.41 (0.62, 3.25) <b>P = 0.31</b>  <u>Model 2:</u> 1.46 (0.57, 3.77) <b>P = 0.33</b>  <u>Model 3:</u> 1.48 (0.63, 3.47) <b>P = 0.27</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Public funding	<p><b>Excluded:</b> individuals who changed schools at baseline, were in special education classes, were in grades other than 6<sup>th</sup> or 7<sup>th</sup> or didn't complete the English-language version of the questionnaire; incomplete data; implausible energy intakes (<math>\leq 20,90</math> KJ or <math>\geq 29,260</math> KJ).</p> <p><b>Follow-up rate:</b> 84%</p> <p><b>n</b> = 548  <b>Sex:</b> 48% female  <b>Ethnicity:</b> 64% white, 15% Hispanic, 14% Afro-American, 8% Asian, 8% American Indian or other  <b>Age:</b> 11-12 y</p> <p><b>Excluding obese at baseline</b> (n=150)  <b>n</b> = 398</p>	on both BMI and triceps-skinfold thickness <sup>385</sup> <sup>th</sup> percentile of age-specific and sex-specific reference data.	<b>Exposure assessment:</b> SFFQ		<b>Model 3:</b> model 2 + total energy intake	<p><b>OR (95%CI) per each serving increase between baseline and follow-up</b></p> <p><u>Model 1:</u> 1.39 (0.99, 1.95)  <b>P = 0.05</b></p> <p><u>Model 2:</u> 1.44 (1.22, 1.70)  <b>P = 0.004</b></p> <p><u>Model 3:</u> 1.60 (1.14, 2.24)  <b>P = 0.02</b></p> <p><b>Baseline intake of ASB was not associated to obesity incidence (p = 0.69). Change in ASB intake from baseline to follow-up was negatively associated with incidence of obesity, OR (95%CI) 0.44 (NR), p = 0.03.</b></p> <p>Results also reported for continuous outcome BMI</p>
<b>Exposure: SSSD + SSFD + TFI</b>							
<b>2</b>	<b>Generation R</b>  The Netherlands	<p><b>N</b> = 9,749</p> <p><b>Population sampled:</b> General population</p>	<b>Incidence of overweight or obesity</b> Weight and height were measured (without shoes and heavy clothing) using an electronic scale and stadiometer.	<p><b>servings/week (median)<sup>16</sup></b></p> <p><b>T1(ref):</b> 3  <b>T2:</b> 8  <b>T3:</b> 15  <b>n</b>  <b>Females</b></p>	NR	<p><b>Model 1:</b> age</p> <p><b>Model 2:</b> model 1 + gestational age at birth, birth weight (SDS), age of mother and father, net household income, maternal BMI, education, smoking, folic acid use, pre-</p>	<p><b>Females</b></p> <p><b>Model 1; OR (95%CI)</b></p> <p>T1(ref): 1  T2: 1.16 (0.74, 1.82)  T3: 1.40 (0.89, 2.20)  <b>P per trend = 0.15</b></p>

<sup>16</sup> Standardised by energy using the residual method

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Leermakers et al. (2015)  5 y  Mixed funding	<p><b>Excluded:</b> Children without information on sugar-containing beverage intake at 13 months or BMI at any time point.</p> <p><b>n</b> = 2,371 Females: 1,188 Males: 1,183</p> <p><b>Ethnicity:</b> Caucasian</p> <p><b>Age Median (IQR)</b> 1.08 (0.98 - 1.18) y</p>	<p>For BMI, age- and sex-specific s.d. scores were obtained using Dutch reference growth curves.</p> <p>Children were classified as overweight according to age- and sex-specific cut-off points from the International Obesity Task Force.</p>	<p><u>T1</u>: 394 <u>T2</u>: 399 <u>T3</u>: 395 <b>Males</b> <u>T1</u>: 392 <u>T2</u>: 393 <u>T3</u>: 398</p> <p><b>Serving size:</b> 150 ml</p> <p><b>Exposure assessment:</b> SFFQ</p>		<p>pregnancy and pregnancy related comorbidities, child hospitalization in first year of life and history of allergy to cow's milk.</p> <p><b>Model 3:</b> model 2 + child's intake of sugar, confectionary, cakes and pastry, breastfeeding, time of introduction of complementary feeding, total energy intake and hours of TV watching.</p>	<p><b>Model 2; OR (95%CI)</b> T1(ref): 1 T2: 1.08 (0.66, 1.76) T3: 1.22 (0.75, 1.99) <b>P per trend = 0.42</b></p> <p><b>Model 3; OR (95%CI)</b> T1(ref): 1 T2: 1.09 (0.67, 1.78) T3: 1.27 (0.78, 2.06) <b>P per trend = 0.34</b></p> <p><b>Males</b></p> <p><b>Model 1; OR (95%CI)</b> T1(ref): 1 T2: 1.08 (0.62, 1.89) T3: 1.00 (0.55, 1.82) <b>P per trend = 0.99</b></p> <p><b>Model 2; OR (95%CI)</b> T1(ref): 1 T2: 1.04 (0.59, 1.82) T3: 0.90 (0.47, 1.72) <b>P per trend = 0.73</b></p> <p><b>Model 3; OR (95%CI)</b> T1(ref): 1 T2: 1.03 (0.57, 1.88) T3: 0.90 (0.44, 1.85) <b>P per trend = 0.75</b></p> <p>Results also reported for continuous outcome BMIZ and percentage of fat mass</p>
3	Amsterdam  The Netherlands	N = 226	<b>Incidence of overweight or obesity</b> BMI was calculated from self-reported weight and	<b>E% [mean (SD)] from sugar-containing</b>	20 (16.7%)	<b>Model 1:</b> crude	<b>OR (95%CI) per each 1E% from sugar-containing beverages</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Weijjs et al. (2011)  8 y  Public funding	<b>Population sampled:</b> General population  <b>Excluded:</b> no explicit permission to be approached again after initial contact.  <b>Loss of follow-up</b> = 101  <b>n</b> = 120 <b>Sex:</b> 46.67% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 4-13 mo	height. BMI standard deviation score (BMIs) was used.  WHO BMIs cut-off point of +1 and +2 were used to define overweight and obesity, respectively.  No data on how self-reported height and weight related to measured height and weight.	<b>beverages at baseline</b>  All = 5.2 (6.3)  Consumers only = 8.7 (6.0)  <b>Exposure assessment:</b> 2-d food record		<b>Model 2:</b> sex, infant age, infant body weight, breastfed at time of assessment, SES  <b>Model 3:</b> animal protein  <b>Model 4:</b> model 2 + model 3	<b>Model 1; OR (95%CI)</b> 1.10 (1.02, 1.18) <b>P</b> = 0.009  <b>Model 2; OR (95%CI)</b> 1.10 (1.02, 1.20) <b>P</b> = 0.021  <b>Model 3; OR (95%CI)</b> 1.11 (1.03, 1.20) <b>P</b> = 0.005  <b>Model 4; OR (95%CI)</b> 1.13 (1.03, 1.24) <b>P</b> = 0.009  Results also reported for continuous outcome BMIz
<b>Exposure: SSSD + SSFD + SSFJ</b>							
3	<b>ELEMENT</b>  Mexico  Cantoral et al. (2015)*  up to 13 y  Public funding	<b>N</b> = 1,079  <b>Population sampled:</b> General population  <b>Excluded:</b> missing information on socio-demographic, dietetic, anthropometric and/or physical activity variables, obesity at baseline (1-5 years)  <b>n</b> = 227  <b>Sex:</b> 54% females <b>Ethnicity:</b> Hispanics <b>Age:</b> 1 y	<b>Incidence of obesity</b> Weight and height were obtained using standardized procedures by trained personnel: weight was measured with a Bame scale rounded to the nearest 0.1 kg and height was recorded with a stadiometer to the nearest 0.1 cm.  These were used to calculate BMI and participants were classified as "obese" according to the WHO criteria (>2SD of the z-score for BMI).	<b>Cumulative intake during pre-school (1-5 y) (range)</b>  <u>Q1 (ref):</u> 1,642-15,242 ml <u>Q2:</u> 15,410-22,484 ml <u>Q3:</u> 22,731-55,913 ml  <b>n</b> <u>Q1 (ref):</u> 78 <u>Q2:</u> 74 <u>Q3:</u> 75  <b>Exposure assessment:</b> SFFQ	<u>Q1 (ref):</u> 15 <u>Q2:</u> 13 <u>Q3:</u> 29	<b>Model 1:</b> crude model  <b>Model 2:</b> concurrent age, sex, breastfeeding up to age 12mo, maternal obesity (at 12mo post-partum), non-SSSD-energy intake, physical activity, TV watching	<b>Model 1; OR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.84 (0.34, 2.02) <u>Q3:</u> 2.69 (1.25, 5.79)  <b>Model 2; OR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.84 (0.32, 2.13) <u>Q3:</u> 2.99 (1.27, 7.00)

ASB, artificially sweetened beverages; BMI, body mass index; CI, confidence interval; cm, centimetre; CVD, cardiovascular disease; d, day; FFQ, food frequency questionnaire; g, grams; HR, hazard ratio; kcal, kilocalories; kg, kilogram; kj, kilojoules; m, metre; mg, milligrams; ml, millilitre; mm, millimetres; mo, month; n, participants analysed; N, participants included in the cohort; NHANES, National

Health and Nutrition Examination Survey; NR, not reported; NS, not significant; OR, odds ratio; oz, ounces; SD, standard deviation; SE, standard error; SES, socioeconomic status; SFFQ, semiquantitative food frequency questionnaire; SSFD, sugar-sweetened fruit drinks; SSFJ, sugar-sweetened fruit juices; SSSD, sugar-sweetened soft drinks; TFJ, total fruit juices; USA, United States of America; WHO, World Health Organization; wk, week; y, years. \*Data provided by the authors. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Incidence of abdominal obesity

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: SSSD</b>							
<b>1</b>	<b>Girona</b>  Spain  Funtikova et al. (2015)  10 y  Public funding	<b>N</b> = 3,058  <b>Population sampled:</b> General population  <b>Excluded:</b> missing data for WC, smoking status, abdominal obesity at baseline  <b>Follow-up rate:</b> 80.3%  <b>n</b> = 1,479  <b>Sex:</b> 49% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 25-74 y	Measured WC midway between the lowest rib and the iliac crest, with participants lying horizontally, and measurement rounded to the nearest 0.5cm.  <b>Abdominal obesity</b> defined by sex-specific cut-offs: >102 cm for men and >88 cm for women.	<b>mL/d (range)</b> <b>C1 (ref):</b> non-consumers <b>C2:</b> >0 and <200 <b>C3:</b> ≥200  <b>n/person years per category of exposure NR</b>  <b>Exposure assessment:</b> SFFQ	336  <b>Cases per category of exposure NR</b>	<b>Model:</b> sex, age, baseline WC, smoking, energy intake, education, physical activity, modified Mediterranean diet score and energy under- and over- reporting	<b>OR (95%CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.22 (0.90, 1.66) <b>C3:</b> 1.77 (1.07, 2.93)  <b>RR (95%CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.18 (0.94, 1.47) <b>C3:</b> 1.48 (1.01, 2.05)  Results also reported for continuous outcome WC
<b>2</b>	<b>KoGES</b>  South Korea  Kang and Kim (2017)  5.7 y (mean)  Public funding	<b>N</b> = 10,030  <b>Population sampled:</b> general population living in Ansan (urban) and Ansong (rural) areas  <b>Excluded:</b> participants who refused to participate in follow-up examinations, insufficient information, non-responders to dietary examination and prevalence of abdominal obesity, CVD or cancer	WC measurements were repeated three times, and then averaged after measuring to the nearest 0.1 cm at the narrowest point between the lowest rib and the right iliac crest.  <b>Abdominal obesity:</b> ≥ 90 cm for men or ≥80 cm for women	<b>Servings/week (range)</b> <b>C1(ref):</b> Rarely or never <b>C2:</b> <1 <b>C3:</b> ≥1 to <4 <b>C4:</b> ≥4  <b>n females</b> <b>C1:</b> 993 <b>C2:</b> 646 <b>C3:</b> 206 <b>C4:</b> 29	<b>Females</b> <b>C1:</b> 405 <b>C2:</b> 254 <b>C3:</b> 82 <b>C4:</b> 15  <b>Males</b> <b>C1:</b> 278 <b>C2:</b> 273 <b>C3:</b> 167 <b>C4:</b> 28	<b>Model 1:</b> age  <b>Model 2:</b> age, income level, education level, alcohol consumption, smoking status, physical activity, BMI, energy intake, percentage of fat, fibre intake and the presence of diseases	<b>OR (95% CI)</b>  <b>Females</b> <b>Model 1 :</b> <b>C1 (ref):</b> 1 <b>C2:</b> 0.96 (0.82, 1.12) <b>C3:</b> 1.11 (0.87, 1.41) <b>C4:</b> 1.78 (1.06, 2.99) <b>P for trend = 0.25</b>  <b>Model 2 :</b> <b>C1 (ref):</b> 1 <b>C2:</b> 0.95 (0.81, 1.11) <b>C3:</b> 1.12 (0.88, 1.43) <b>C4:</b> 1.32 (0.78, 2.23)



RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		<b>Follow-up rate:</b> 63.3 %  <b>n=</b> 5,012 females: 1,874 males: 3,138  <b>Ethnicity:</b> Asian  <b>Age:</b> 40-69 y		<b>n</b> <b>males</b> C1: 1,127 C2: 1,237 C3: 665 C4: 109  <b>Serving size:</b> 200 ml  <b>Exposure assessment:</b> SFFQ			<b>P for trend = 0.44</b>  <b>Males</b> <b>Model 1 :</b> C1 (ref): 1 C2: 0.84 (0.71, 0.99) C3: 1.08 (0.89, 1.31) C4: 1.11 (0.75, 1.65) <b>P for trend = 0.98</b>  <b>Model 2 :</b> C1 (ref): 1 C2: 0.84 (0.73, 1.03) C3: 1.07 (0.87, 1.31) C4: 1.11 (0.74, 1.65) <b>P for trend = 0.95</b>
<b>Exposure: SSSD + SSFD</b>							
<b>1</b>	<b>CARDIA</b>  USA  Duffey et al. (2010)  20 y  Mixed funding	<b>N =</b> 5,115  <b>Population sampled:</b> general population of 4 centres selected to balance subgroups of race, sex, education and age  <b>Excluded:</b> pregnant women, individuals with the abdominal obesity at years 0 or 7  <b>n =</b> 2,444 <b>Sex:</b> 53.5% females <b>Ethnicity:</b> Caucasian 52.6%, Black 47.4% <b>Age:</b> 18-30 y	Waist circumference was measured as the average of 2 measures at the minimum abdominal girth (nearest 0.5cm) from participants standing upright. <b>Abdominal obesity defined as WC</b> > 88cm for women or >102cm for men.	<b>Kcal/day (mean±SEM)</b>  <b>Year 0;</b> n=5,034 167±3  <b>Year 7;</b> n= 3,877 196±8  Average of intake at years 0 and 7 was used for the analysis = NR  <b>Exposure assessment:</b> SFFQ	637	<b>Model:</b> race, gender, centre, age, weight, smoking status, energy from food, total physical activity, energy from other beverages (low-fat milk, whole-fat milk and fruit juice), and energy from alcohol	<b>Per 100kcal/d increase</b> <b>RR (95% CI)</b> 1.06 (1.02, 1.10) <b>P &lt; 0.05</b>
<b>Exposure: SSSD + SSFD + TFJ</b>							
<b>3</b>	<b>TLGS</b>  Iran	<b>N=</b> 15,005	Waist circumference was measured at the umbilicus using a measuring tape, without pressure to body	<b>mL/d (median)</b> <b>Q1 (ref):</b> 9.3 <b>Q2:</b> 32.0 <b>Q3:</b> 58.6	NR	<b>Model 1:</b> baseline age, sex, total energy intake, physical activity and family history of diabetes	<b>Model 1; OR (95%CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 1.53 (0.63, 3.71) <b>Q3:</b> 1.65 (0.61, 3.94)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Mirmiran et al. (2015)  3.6 y (mean)  Public funding	<p><b>Population sampled:</b> general population from one district of Tehran</p> <p><b>Excluded:</b> incomplete dietary intakes or missing measures of MetS components, reported energy intakes to energy requirements ratio beyond <math>\pm 3SD</math>; abdominal obesity at baseline (survey 3).</p> <p><b>Follow-up rate:</b> 86%</p> <p><b>n</b> = 327  <b>Sex:</b> 68 % females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 6-18 y</p>	<p>surfaces, and was recorded to the nearest 0.5cm.</p> <p><b>Abdominal obesity</b> was defined as <math>WC \geq 91</math> and <math>\geq 89cm</math> for men and women, respectively, during follow-up (survey 4).</p>	<p>Q4: 142.2</p> <p><b>N of subjects per quartile for this outcome NR</b></p> <p><b>Exposure assessment:</b> SFFQ</p>		<p><b>Model 2:</b> model 1 + dietary fibre, tea and coffee, red and processed meat, fruit and vegetables</p> <p><b>Model 3:</b> model 2 + BMI</p>	<p>Q4: 2.94 (1.27, 6.82)  <b>P per trend: 0.012</b></p> <p><b>Model 2; OR (95%CI)</b>  Q1 (ref): 1  Q2: 1.58 (0.65, 3.86)  Q3: 1.70 (0.70, 4.09)  Q4: 2.97 (1.23, 7.19)  <b>P per trend: 0.017</b></p> <p><b>Model 3; OR (95%CI)</b>  Q1 (ref): 1  Q2: 2.16 (0.82, 5.68)  Q3: 1.86 (0.71, 4.84)  Q4: 3.66 (1.40, 9.59)  <b>P per trend: 0.016</b></p>
<b>Exposure: SSSD + SSFD + SSFJ</b>							
3	<p><b>ELEMENT</b></p> <p>Mexico</p> <p>Cantoral et al. (2015)*</p> <p>up to 13 y</p> <p>Public funding</p>	<p><b>N</b> = 1,079</p> <p><b>Population sampled:</b> General population</p> <p><b>Excluded:</b> missing information on socio-demographic, dietetic, anthropometric or physical activity variables, abdominal obesity at baseline (1-5 years)</p> <p><b>n</b> = 227  <b>Sex:</b> 54% females  <b>Age:</b> 1 y</p>	<p>Waist circumference was obtained using standardized procedures by trained personnel, it was measured using a measuring tape to the nearest 0.1cm.</p> <p>Waist circumference <math>\geq 90^{th}</math> percentile for age and sex was used to define <b>abdominal obesity</b>.</p>	<p><b>Cumulative intake during pre-school 1-5 y (range)</b></p> <p>Q1 (ref): 1,642-15,242ml  Q2: 15,410-22,484ml  Q3: 22,731-55,913ml</p> <p><b>n</b>  Q1 (ref): 78  Q2: 74  Q3: 75</p> <p><b>Exposure assessment:</b> SFFQ</p>	<p>Q1 (ref): 13  Q2: 14  Q3: 22</p>	<p><b>Model 1:</b> crude model</p> <p><b>Model 2:</b> child sex, breastfeeding up to age 12mo, maternal obesity (at 12mo post-partum), concurrent age, non-SSSD-energy intake, physical activity, TV watching</p>	<p><b>Model 1; OR (95%CI)</b>  Q1 (ref): 1  Q2: 1.15 (0.47, 2.80)  Q3: 2.29 (1.01, 5.19)</p> <p><b>Model 2; OR (95%CI)</b>  Q1 (ref): 1  Q2: 1.14 (0.42, 3.07)  Q3: 2.70 (1.03, 7.03)</p>
<b>Exposure: 100% FJ</b>							
1	<p><b>CARDIA</b></p> <p>USA</p>	<p><b>Same population and exclusion criteria as for SSSD+SSFD</b></p>	<p><b>Same ascertainment of outcome as for SSSD+SSFD</b></p>	<p><b>Kcal/day (mean<math>\pm</math>SEM)</b></p>	637	<p><b>Model:</b> race, gender, centre, age, weight, smoking status, energy from food, total physical activity, energy from other</p>	<p><b>Per 100kcal/d increase RR (95% CI)</b></p>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Duffey et al. (2010)*  20 y  Mixed funding			<b>Year 0;</b> n=5,034 115±2  <b>Year 7;</b> n= 3,877 114±9  Average of intake at 0 and 7 years used for the analysis = NR  <b>Exposure assessment:</b> SFFQ		beverages (low-fat milk, whole-fat milk and SSBs), and energy from alcohol	0.98 (0.90, 1.06)
<b>1</b>	<b>Girona</b>  Spain  Funtikova et al. (2015)  10 y  Public funding	<b>Same exclusion criteria as for SSSD</b>	<b>Same ascertainment of outcome as for SSSD</b>	<b>mL/d (range)</b> <b>C1 (ref):</b> non-consumers <b>C2:</b> >0 and <200 <b>C3:</b> ≥200  <b>n/person years per category of exposure NR</b>  <b>Exposure assessment:</b> SFFQ	336  <b>Cases per category of exposure NR</b>	<b>Model:</b> sex, age, baseline WC, smoking, energy intake, education, physical activity, modified Mediterranean diet score and energy under- and over- reporting.	<b>OR (95%CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 0.98 (0.72, 1.31) <b>C3:</b> 0.74 (0.49, 1.13)  <b>RR (95%CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.00 (0.80, 1.24) <b>C3:</b> 0.82 (0.72, 1.12)  Results also reported for continuous outcome WC

BMI, body mass index; CI, confidence interval; cm, centimetre; d, day; FJ, fruit juice; HR, hazard ratio; kcal, kilocalories; MetS, metabolic syndrome; ml, millilitres; mo, month; n, participants analysed; N, participants included in the cohort; NR, not reported; OR, odds ratio; RR, risk ratio; SD, standard deviation; SEM, standard error mean; SFFQ, semiquantitative food frequency questionnaire; SSB, sugar-sweetened beverages; SSFD, sugar-sweetened fruit drinks; SSFJ, sugar-sweetened fruit juices; SSSD, sugar-sweetened soft drinks; TFJ, total fruit juices; USA, United States of America; WC, waist circumference; wk, week; y, years. \*Data provided by the authors. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Ectopic fat deposition

### Liver fat

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups Exposure assessment method	Outcome	Model covariates	Results	
Exposure: total sugars								
1	ALSPAC  UK  Anderson et al. (2015)  14 y  Mixed funding	N = 15,247  <b>Population sampled:</b> General population living within a defined part of the country  <b>Excluded:</b> no ultrasound scans (USS) at follow-up (17-18 y), no measure of dietary intake between 3 and 13 years, known history of jaundice or hepatitis, taking medication that would indicate hepatic disease, taking medication known to influence liver function, regular alcohol drinking.  n = 1,786 USS liver fat: 1,632 USS liver stiffness: 1,638  <b>Sex:</b> 58.1% females <b>Ethnicity:</b> Caucasian <b>Aae:</b> 3 y	<b>Liver fat (surrogate for NAFLD)</b>  USS: echogenicity recorded as present or absent. Level of agreement among the 4 sonographers 98%.  <b>Liver stiffness (surrogate for liver fibrosis)</b>  USS: acoustic radiation force impulse measured as shear velocity in meters per second using standard protocols	<b>g/d †</b>  3 y: NR 7 y: NR 13 y: NR  <b>n</b> USS liver fat: 1,632 USS liver stiffness: 1,638  <b>Exposure assessment:</b> at last one 3-day food diary and/or SFFQ	<b>Liver fat</b>  Present: 2.8% Absent: 97.2%  <b>Liver stiffness</b> (median (IQR))  1.2 (1.1, 1.3) m/s	<b>Model 1:</b> energy intake  <b>Model 2:</b> model 1 + sex, age at outcome assessment, maternal pre-pregnancy BMI, maternal age, social class, maternal education and parity  <b>Model 3:</b> Model 2 + total body fat at the time of outcome assessment	<b>Exposure at 3, 7 and 10 y of age</b>  <b>Liver fat at 17-18 y</b> Per each 10g/d increase  <b>Model 1; OR (95%CI)</b> 3 y: 1.29 (0.82, 2.03) 7 y: 1.09 (0.83, 1.43) 13 y: 0.90 (0.72, 1.13)  <b>Model 2; OR (95%CI)</b> 3 y: 1.26 (0.80, 1.98) 7 y: 1.12 (0.85, 1.47) 13 y: 0.96 (0.77, 1.22)  <b>Model 3; OR (95%CI)</b> 3 y: 1.50 (0.92, 2.45) 7 y: 1.32 (0.98, 1.78) 13 y: 1.13 (0.89, 1.43)  <b>Liver stiffness at 17-18 y</b> Per each 10g/d increase  <b>Model 1; %Δ (95%CI)</b> 3 y: 1 (-1, 2) 7 y: 0 (-1, 1) 13 y: 0 (-1, 1)  <b>Model 2; %Δ (95%CI)</b> 3 y: 1 (-1, 2) 7 y: 0 (-1, 1) 13 y: 0 (-1, 1)  <b>Model 3; %Δ (95%CI)</b> 3 y: 1 (0, 2) 7 y: 0 (0, 1) 13 y: 0 (0, 1)	

† Exposure adjusted for total energy intake using the nutrient residuals model

## Visceral adipose tissue

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups Exposure assessment method	Outcome	Model covariates	Results		
Exposure: SSSD+SSFD									
1	Framingham-3Gen	N = 4,095  <b>Population sampled:</b> General population  <b>Excluded:</b> not being eligible for CT scans (weight > 160 kg, women < 40 y, men <35 y), missing CT scan at baseline or follow-up, missing data on exposure or covariates, bariatric surgery, history of CVD or cancer.  <b>n</b> = 1,003  <b>Sex:</b> 45% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 45.3 y (mean)	VAT and VAT:SAAT ratio  CT scanning obtained 25 contiguous slices covering 125mm superiorly from the upper edge of vertebrae S1. VAT and SAAT were separated manually tracing the abdominal muscular wall. Intra-class correlations for VAT and SAAT readings > 0.99.	Servings/week (median) C1: 0 C2: 0.5 C3: 3 C4: 11  <b>Serving size</b> = 12 oz (355mL)  <b>n</b> C1: 317 C2: 196 C3: 356 C4: 134  <b>Exposure assessment:</b> SFFQ	Change in VAT and VAT:SAAT ratio from baseline to follow-up  Baseline VAT (cm <sup>3</sup> ) Mean ± SD: C1: 1454 ± 902 C2: 1322 ± 868 C3: 1731 ± 896 C4: 1771 ± 831  Baseline VAT:SAAT ratio (geometric mean ± SD): C1: 0.44 ± 0.30 C2: 0.47 ± 0.33 C3: 0.62 ± 0.43 C4: 0.72 ± 0.39	Model 1: baseline outcome values, sex, age, smoking, physical activity, energy intake, alcohol intake, saturated fat intake, diet soda intake, multivitamin use, and intake of whole grain, fruit, vegetable, coffee, nuts, and fish  Model 2: model 1 + change in body weight	Exposure: Baseline  Change in VAT volume (cm <sup>3</sup> )  Model 1; mean (95%CI) C1: 659 (582, 735) C2: 675 (582, 767) C3: 709 (640, 777) C4: 809 (683, 935) P per trend 0.06  Model 2; mean (95%CI) C1: 658 (602, 713) C2: 649 (582, 716) C3: 707 (657, 757) C4: 852 (760, 943) P per trend <0.001	Exposure: Baseline  Change in VAT:SAAT ratio  Model 1; mean (95%CI) C1: 0.09 (0.06, 0.11) C2: 0.08 (0.05, 0.11) C3: 0.12 (0.10, 0.14) C4: 0.15 (0.11, 0.18) P per trend 0.007  Model 2; mean (95%CI) C1: 0.09 (0.07, 0.11) C2: 0.08 (0.05, 0.10) C3: 0.12 (0.10, 0.14) C4: 0.15 (0.11, 0.19) P per trend 0.004	
								No association observed for ASBs	

ASSD, artificially sweetened soft drink; BMI, body mass index; CI, confidence interval; cm, centimetre; CT, computed tomography; CVD, cardiovascular disease; d, day; g, gram; IQR, interquartile range; kg, kilogram; ml, millilitre; mm, millimetre; mo, month; n, participants analysed; N, participants included in the cohort; NALFD, non-alcoholic fatty liver disease; OR, odds ratio; oz, ounces; SAAT, subcutaneous abdominal adipose tissue; SFFQ, semiquantitative food frequency questionnaire; SSFD, sugar-sweetened fruit drinks; SSSD, sugar-sweetened soft drinks; UK, United Kingdom; USA, United States of America; USS, ultrasound scans; VAT, visceral adipose tissue; wk, week; y, year. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Continuous measures of glucose homeostasis

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
<b>Exposure: total sugars</b>							
<b>2</b>	<b>Seven Countries</b>  The Netherlands, West Finland and East Finland  Feskens et al. (1995)  20 y  Public funding	<b>N</b> = 2,589  <b>Population sampled:</b> General population  <b>Excluded:</b> treated diabetes or death at follow-up, incomplete anthropometric and/or dietary data at baseline. Only a random sample invited to the last follow-up  <b>n</b> = 338  <b>Sex:</b> Males  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 50 – 70 y	<b>OGTT 2-h glucose concentration</b>  <b>OGTT</b> was carried out at end of follow-up according to the WHO guidelines <sup>17</sup> . The first blood sample was obtained in the morning after an overnight fast. The second sample was obtained 2 h after the glucose load of 75 g. Samples were collected in tubes with sodium fluoride. In Finland, plasma glucose was determined using the glucose dehydrogenase method and the hexokinase method was used in the Netherlands.	<b>%E (age and cohort adjusted means)</b> <b>NR</b> for pooled cohort  <b>Baseline</b> <b>NGT:</b> 24.7 <b>IGT:</b> 25  <b>At Follow-up:</b> <b>NGT:</b> 24.2 <b>IGT:</b> 26  <b>Method:</b> Dietary history	<b>Total sugars</b> intake at baseline and changes in total sugars intake over the 20-y follow-up vs OGTT 2-h glucose concentration at end follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> cohort, age, baseline BMI, and baseline energy intake (+ baseline intake of total sugars for change in intake during follow-up analysis)	Non-significant (negative) association between baseline total sugar intake and OGTT 2-h glucose concentrations at end follow-up. The association for changes in total sugar intake was also non-significant, but positive.  <b>Exposure: Baseline</b> <b>Per each 1 %E increase</b> <b>β coefficient ± SE</b> -0.014 ± 0.032, <b>NS</b>  <b>Exposure: Change from baseline</b> <b>Per each 1 %E increase</b> <b>β coefficient ± SE</b> 0.014 ± 0.025, <b>NS</b>
<b>Exposure: free and/or added sugars</b>							
<b>1</b>	<b>DONALD</b>  Germany	<b>N</b> = >1,300  <b>Population sampled:</b> General population from Dortmund	<b>HOMA-IR</b>  Venous blood samples were drawn after an overnight fast. Fasting	<b>E%<sup>19</sup> (means)</b> T1: 13.1 T2: 14.2 T3: 17	<b>Free sugars</b> intake at baseline vs HOMA-IR at end of follow-up	<b>Model 1:</b> sex, age and energy (residuals †)	Free sugars intake at baseline, from all sources or from liquids only, was not significantly associated with HOMA-IR at end of follow-up.

<sup>17</sup> World Health Organization: Diabetes Mellitus: Report on a WHO Study Group. Geneva, World Health Org., 1985 (Tech. Rep. Ser., no. 727).

<sup>19</sup> Baseline added sugar (% energy) by tertiles of dietary glycaemic index

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
	Goletzke et al. (2013b)  12.6 y (mean)  Public funding	<b>Excluded:</b> consistently underreported energy intake, missing anthropometric measurements in adolescence or adulthood, missing data on dietary intake or covariates  <b>n=</b> 226  <b>Sex:</b> 53.5% Females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> Females 9-14 y Males: 10-15 y	blood samples were used to calculate <b>HOMA-IR</b> . <sup>18</sup>	<b>from liquid sources</b> T1: 3.21 T2: 4.03 T3: 6.07  <b>n</b> T1: 75 T2: 76 T3: 75  <b>Method:</b> 3-d DR	<b>Data collection:</b> annually until end of follow-up	<b>Model 2:</b> model 1 + early life factors (first born), BMI SDs at baseline, maternal education, and fibre and protein  <b>Model 3:</b> model 2 + waist circumference in younger adulthood	No prospective association was observed between free sugars and HOMA-β (data not shown)
							<b>Means (95% CI)</b>  <b>All sources</b> <u>Model 1</u> T1: 2.61 (2.39, 2.86) T2: 2.64 (2.41, 2.89) T3: 2.48 (2.26, 2.71) <b>P for trend = 0.7</b>  <u>Model 2</u> T1: 2.57 (2.32, 2.86) T2: 2.57 (2.34, 2.82) T3: 2.29 (2.07, 2.54) <b>P for trend = 0.3</b>  <u>Model 3</u> T1: 2.53 (2.29, 2.80) T2: 2.56 (2.35, 2.80) T3: 2.33 (2.11, 2.57) <b>P for trend = 0.4</b>

<sup>18</sup> Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985;28:412–419.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
2	<b>QUALITY</b>	<b>N = 630</b>  <b>Population sampled:</b> General population from Quebec with at least one biological parent that had obesity and/or abdominal obesity  <b>Excluded:</b> Diabetes, following a very restricted diet (< 2510 kJ/d), regular medication use, and serious psychological ailments.  <b>n= 457</b> <b>n for Matsuda-ISI = 419</b>  <b>Follow-up rate:</b> 97%  <b>Sex:</b> 44.5% females  <b>Ethnicity:</b> Caucasian  <b>Age:</b> 8 – 10 y	<b><u>FG, FI, HOMA-IR and Matsuda-ISI</u></b>  Blood samples were obtained after an overnight fast. <b>OGTT</b> - blood was collected at 30, 60, 90, and 120 min after an oral glucose dose of 1.75 g/kg body weight (up to a maximum of 75 g). <b>HOMA-IR</b> was calculated using the formula: fasting plasma glucose (mmol/L) * fasting plasma insulin (pmol/L)/22.5. <b>Matsuda-ISI</b> was calculated as 10,000/square root [(fasting plasma glucose * fasting plasma insulin) * (mean OGTT) glucose 3 mean OGTT insulin)].	<b>g/d from solid sources</b> <b>Mean ± SD</b> 40.4 ± 22.2  <b>g/d from liquid sources</b> 11.4 ± 12.5  <b>Exposure assessment:</b> Three 24-h DR	<b>Added sugars from liquid and solid sources</b> at baseline vs changes in FG, FI, HOMA-IR and Matsuda-ISI  <b>Data collection:</b> exposure at baseline, outcome at baseline and end of follow-up	<b>Model:</b> baseline level of outcome variable, age, sex, tanner stage, energy intake, fat mass index, and physical activity.	<b>Significant positive</b> associations between baseline intake of added sugars from liquid sources and changes in FG, FI and HOMA-IR over follow-up. Associations were also positive for added sugars from solids, but non-significant. Associations with changes in Matsuda-ISI were <b>significantly negative</b> for added sugars from liquid sources and non-significant (negative) for added sugars from solid sources.  <b>Per each 10 g/d increase β coefficients (95% CI)</b>  <b><u>Solid sources</u></b> <u>FG (mmol/L):</u> 0.001 (-0.016, 0.018) <u>FI (rmol/L):</u> 0.196 (-0.904, 1.296) <u>HOMA-IR:</u> 0.007 (-0.033, 0.047) <u>Matsuda-ISI:</u> -0.036 (-0.227, 0.156)  <b><u>Liquid sources</u></b> <u>FG (mmol/L):</u> 0.039 (0.015, 0.063) <b>P &lt; 0.01</b> <u>FI (rmol/L):</u> 2.261 (0.676, 3.845) <b>P &lt; 0.01</b> <u>HOMA-IR:</u> 0.091 (0.034, 0.149) <b>P &lt; 0.01</b> <u>Matsuda-ISI:</u> -0.356 (-0.628, -0.084) <b>P &lt; 0.01</b>
	<b>Exposure: sucrose</b>						
2	<b>CARDIA</b>	<b>N = 5,115</b>  <b>Population sampled:</b> general population of 4 centres selected to balance subgroups of race, sex, education and age	<b><u>FI</u></b>  Blood was drawn from participants after a 12 hour fast. <b>FI</b> was measured at baseline examination by a nonspecific insulin assay. At follow-up	<b>% E</b> NR  <b>Method:</b> SFFQ	<b>Changes in sucrose</b> intake vs changes in FI over the 7-y follow-up  <b>Data collection:</b>	<b>Model:</b> baseline intake of sugars, age, and time period	<b>Significant negative</b> association between changes in sucrose intake and changes in fasting insulin over the 7-y follow-up in white females only.  <b>Per each SD (6%E) increase Mean change (SD) (μU/ml)</b> Black females: 0.1 White females: -0.7, <b>p &lt; 0.05</b>



RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
	Public funding	<b>Excluded:</b> fasting <10h before examination, pregnancy at time of examination, diabetes, missing insulin values using the specific insulin assay, extreme insulin values  <b>n=</b> 3,095 Black females: 770 White females: 839 Black males: 612 White males: 874 <b>Age:</b> 18 – 30 y	examination radioimmunoassay was employed. To ensure comparability, FI was measured in Year 7 participants on sera stored one year from the Year 7 examination, and also used the new assay on sera stored for 8 years from Year 0.		baseline and end of follow-up		<u>Black males:</u> -0.0 <u>White males:</u> -0.2  <i>Spread values not reported</i>
<b>Exposure: fructose</b>							
3	<b>TLGS</b>  Iran  Bahadoran et al. (2017)  6.7 y (mean)  Public funding	<b>N</b> = 15,005  <b>Population sampled:</b> general population from one district of Tehran  <b>Excluded:</b> Unusual energy intake (<800 kcal/day or >4200 kcal/day, respectively), or were on specific diets for hypertension, diabetes or dyslipidemia; those with a history of CVD at baseline.  <b>n</b> = 904 <b>Follow-up rate:</b> 99.5% <b>Sex:</b> 56.5% females <b>Ethnicity:</b> Caucasian <b>Age (mean ± SD):</b> 38.1 ± 13.3 y	<b>FI and HOMA-IR</b>  Over-night fasting blood samples were collected from all study participants, at baseline and again at the follow-up examination. <b>Fasting serum insulin</b> was measured, by electrochemiluminescence immunoassay. <b>HOMA-IR</b> was defined as follows: fasting insulin (μU/mL) * fasting glucose (mmol/L)/22.5.	<b>%E</b> <b>Mean ± SD</b> 6.4 ± 3.7  <b>Method:</b> SFFQ	<b>Fructose</b> intake at baseline vs changes in FI and HOMA-IR over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> age	<b>Significant positive</b> association between fructose intake at baseline and changes in FI and HOMA-IR over the mean follow-up of 6.7 years.  <b>Per each 1 %E increase β coefficients (95% CI)</b> <b>FI:</b> 0.117 (0.023, 0.211) <b>HOMA-IR:</b> 0.024 (0.001, 0.048)
<b>Exposure: SSSD+SSFD</b>							
1	<b>Framingham-Offspring</b>	<b>N</b> = 5,135	<b>HOMA-IR</b>	<b>servings/wk (median)</b> <b>Q1 (ref):</b> 0 <b>Q2:</b> 1	Cumulative average <b>SSSD+SSFD</b>	<b>Model 1:</b> age and sex	<b>Model 1; Geometric means (95% CI)</b> <b>Q1 (ref):</b> 2.94 (2.81, 3.07) <b>Q2:</b> 2.88 (2.75, 3.01)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
	USA  Ma et al. (2016a)  14 y (median)  Public funding	<p><b>Population sampled:</b> offspring of Original Cohort (sampled from the general population of Framingham) and their spouses</p> <p><b>Excluded:</b> not report of beverage exposure, prediabetes or T2DM at baseline, missing prediabetes status at baseline or follow-up, missing data on covariates.</p> <p><b>n</b> = 2,076</p> <p><b>Sex:</b> 59.6 % females</p> <p><b>Age:</b> 30-59 y</p>	<p>Fasting blood samples were collected at baseline and at the end of follow-up examination.</p> <p><b>HOMA-IR</b> was calculated as Fasting insulin (<math>\mu\text{U/mL}</math>) x fasting glucose (<math>\text{mmol/L}</math>) <math>\div 22:5</math></p>	<p><u>Q3:</u> 2 <u>Q4:</u> 6</p> <p>Serving size = 12 fl oz (360 mL)</p> <p><b>n</b> <u>Q1 (ref):</u> 522 <u>Q2:</u> 518 <u>Q3:</u> 518 <u>Q4:</u> 518</p> <p><b>Method:</b> SFFQ</p> <p><i>Cumulative intake (i.e. mean intake reported at examinations up to and including the examination of prediabetes diagnosis)</i></p>	intake vs HOMA-IR at end of follow-up	<p><b>Model 2:</b> model 1 + baseline HOMA-IR, smoking, hypertension, physical activity, BMI, energy intake, alcohol intake, fruit juice intake, diet soda intake, Dietary Guidelines Adherence Index (DGA1) score</p> <p><b>Model 3:</b> model 2 + BMI change</p> <p><b>Model 4:</b> model 2 except DGA1 score was replaced with intake of individual foods including coffee, whole grains, vegetables, red meat, nuts, and fish.</p> <p><b>Model 5:</b> model 4 + BMI change</p> <p><b>Adjustments as specified in Models 4 and 5 did not materially change the geometric means as estimated in Model 1; adjustments as specified in Model 3 did not materially change the RRs as estimated in Model 2 (not shown)</b></p>	<p><u>Q3:</u> 3.00 (2.87, 3.14) <u>Q4:</u> 3.24 (3.10, 3.39) <b>P per trend &lt;0.001</b></p> <p><b>Model 2; Geometric means (95% CI)</b> <u>Q1 (ref):</u> 2.90 (2.79, 3.01) <u>Q2:</u> 2.94 (2.84, 3.05) <u>Q3:</u> 3.07 (2.96, 3.18) <u>Q4:</u> 3.15 (3.02, 3.27) <b>P per trend = 0.006</b></p> <p><b>No association was observed for ASB (P per trend = 0.25)</b></p>
<b>Exposure: SSSD+SSFD+SSFJ</b>							

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
1	<b>WAPCS</b>  Australia  Ambrosini et al. (2013)  3 y  Unclear	<b>N</b> = 2,868  <b>Population sampled:</b> offspring from mothers from the Raine study  <b>Excluded:</b> Subjects who reported not fasting before venepuncture.  <b>n per outcome</b> <b>FG</b> <b>n</b> = 1,124 females= 537 males= 587 <b>FI and HOMA-IR</b> <b>n</b> = 1,083 females= 519 males= 564  <b>Ethnicity:</b> Caucasian <b>Age (mean ± SD):</b> 14.0 ± 0.2 y	<b>FG, FI and HOMA-IR</b>  Blood samples were collected the morning after an overnight fast. <b>HOMA-IR</b> was calculated as Fasting insulin (μU/mL) x fasting glucose (mmol/L) ÷ 22:5.	<b>g/d (range (mean ± SD))</b> <b>T1 (ref):</b> 0 – 130 (48 ± 39) <b>T2:</b> 130 – 329 (223 ± 59) <b>T3:</b> 331 – 2,876 (665 ± 351)  <b>n for those changing tertiles of SSB intake = NR</b>  <b>Method:</b> SFFQ	<b>Changes in SSSD+SSFD+SSFJ intake vs percent of change in FG, FI and HOMA-IR over the 3-y follow-up</b>  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> age, pubertal stage, physical fitness, dietary misreporting, maternal education, and family income  <b>Model 2:</b> Model 1 + BMI  <b>Model 3:</b> Model 2 + Healthy and Western dietary pattern scores	Non-significant (negative) relationship between changes in SSSD+SSFD+SSFJ intake and changes in FG, FI and HOMA-IR over the 3-y follow-up. A positive relationship in the first model becomes negative after adjustment for BMI for all three outcomes.
							<b>Per each tertile of intake increase Δ% (95% CI) vs T1</b>  <b>Females - FG</b> <b>Model 1:</b> <b>T2:</b> 0.4 (-1.0, 1.8) <b>T3:</b> 0.1 (-1.5, 1.6) <b>p for trend 0.88</b>  <b>Model 2:</b> <b>T2:</b> 0.4 (-1.0, 1.9) <b>T3:</b> -0.3 (-1.9, 1.3) <b>p for trend 0.80</b>  <b>Model 3:</b> <b>T2:</b> 0.1 (-1.3, 1.5) <b>T3:</b> -1.2 (-3.0, 0.5) <b>p for trend 0.22</b>  <b>Females - FI</b> <b>Model 1:</b> <b>T2:</b> 3.1 (-4.7, 11.0) <b>T3:</b> 6.1 (-2.4, 14.7) <b>p for trend = 0.15</b>  <b>Model 2:</b> <b>T2:</b> 3.2 (-4.3, 10.7) <b>T3:</b> 0.0 (-8.3, 8.2) <b>p for trend 0.91</b>  <b>Model 3:</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
							<p>T2: 1.1 (-6.6, 8.8) T3: -4.5 (-13.8, 4.9) <b>p for trend 0.42</b></p> <p><b>Females – HOMA-IR</b> <b>Model 1:</b> T2: 2.9 (-13.6, 19.5) T3: 6.1 (-11.9, 24.0) <b>p for trend 0.50</b></p> <p><b>Model 2:</b> T2: 3.2 (-12.4, 18.8) T3: -7.5 (-24.7, 9.6) <b>p for trend 0.46</b></p> <p><b>Model 3:</b> T2: -1.4 (-17.6, 14.7) T3: -18.1 (-37.7, 1.5) <b>p for trend 0.09</b></p> <p><b>Model 2:</b> T2: 0.9 (-7.3, 9.1) T3: -0.3 (-8.4, 7.7) <b>p for trend 0.93</b></p> <p><b>Model 3:</b> T2: 0.2 (-8.2, 8.6) T3: -1.4 (-10.3, 7.4) <b>p for trend 0.74</b></p> <p><b>Males – HOMA-IR</b> <b>Model 1:</b> T2: 6.3 (-12.1, 24.6) T3: 4.8 (-13.5, 23.2) <b>p for trend 0.62</b></p> <p><b>Model 2:</b> T2: -0.5 (-17.2, 16.2) T3: -1.8 (-18.3, 14.7) <b>p for trend 0.83</b></p> <p><b>Model 3:</b> T2: -3.5 (-20.6, 13.6) T3: -7.8 (-25.8, 10.2) <b>p for trend 0.40</b></p>

BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; d, day; DR, dietary record; FG, fasting glucose; FI, fasting insulin; HOMA-IR, Homeostasis model assessment of insulin resistance; IGT, impaired glucose tolerance; kcal, kilocalories; kg, kilograms; kJ, kilojoules; Matsuda-ISI, Matsuda insulin sensitivity index; n, participants analysed; N, participants included in the cohort; NGT, normal glucose tolerance; NR, not reported; NS, non-significant; OGTT, oral glucose tolerance test; SD, standard deviation; SE, standard error; SFFQ, semiquantitative food frequency questionnaire; SSFD, sugar-sweetened fruit drinks; SSFJ, sugar-sweetened fruit juices; SSSD, sugar-sweetened soft drinks; USA, United States of America; y, years. † Exposure adjusted for total energy intake using the nutrient residuals model. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Incidence of Type 2 diabetes mellitus

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: total sugars</b>							
<b>1</b>	<b>FMCHES</b>  Finland  Montonen et al. (2007)  12 y  Public funding	<b>N</b> = 51,522  <b>Population sampled:</b> general population from various regions of Finland  <b>Excluded:</b> no dietary history interview, age <40 or >69 y, reported a daily energy intake of <800 kcal or > 6,000 kcal, T2DM at baseline, missing covariates  <b>n</b> = 4,284 <b>Sex:</b> 47% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 40-69 y	Nationwide registry of patients receiving drug reimbursement for hypoglycaemic agents.  Medical certificates of all the cases were checked and met WHO <sup>20</sup> diagnostic criteria for T2DM.	<b>g/d (median)<sup>†</sup></b> <b>Q1 (ref):</b> 92 <b>Q2:</b> 115 <b>Q3:</b> 136 <b>Q4:</b> 171  <b>n</b> <b>Q1 (ref):</b> 1,066 <b>Q2:</b> 1,068 <b>Q3:</b> 1,075 <b>Q4:</b> 1,075  <b>Exposure assessment:</b> DHI (including SFFQ)	<b>Q1 (ref):</b> 43 <b>Q2:</b> 47 <b>Q3:</b> 37 <b>Q4:</b> 48	<b>Model 1:</b> age, sex, BMI, energy intake, smoking, geographic area, physical activity, family history of diabetes  <b>Model 2:</b> model 1 + prudent dietary pattern score, conservative pattern score  <b>Model 3:</b> model 2 + serum cholesterol, blood pressure, history of infarction, history of angina pectoris and history of cardiac failure  <b>Adjustments as specified in Model 2 did not materially change the RRs as estimated in Models 1 or 3 (not shown)</b>	<b>Model 1; RR (95% CI)</b> <b>Q1:</b> 1 <b>Q2:</b> 1.32 (0.87, 2.01) <b>Q3:</b> 1.07 (0.68, 1.69) <b>Q4:</b> 1.44 (0.93, 2.23) <b>P per trend = 0.18</b>  <b>Model 3; RR (95% CI)</b> <b>Q1:</b> 1 <b>Q2:</b> 1.28 (0.84, 1.95) <b>Q3:</b> 1.12 (0.71, 1.77) <b>Q4:</b> 1.42 (0.90, 2.24) <b>P per trend = 0.20</b>
<b>1</b>	<b>WHS</b>  USA  Janket et al. (2003)*  6 y (median)  Public funding	<b>N</b> = 39,876  <b>Population sampled:</b> health professionals  <b>Excluded:</b> self-reported CHD, stroke, cancer, cases of T2DM at baseline, uncomplete FFQ or reported unreasonable energy intake (<600 or >3,500 kcal/d)  <b>n</b> = 38,480	Self-reported incident cases identified via annual mailed questionnaires plus supplementary questionnaire to all cases asking about the onset of the disease, symptoms, diagnostic tests, and hypoglycemic treatment. Cases ascertained based on the supplementary questionnaire according to the ADA criteria (2003) <sup>21</sup> .  Positive predictive value of incident T2DM = 97.5% as	<b>g/d (median)<sup>†</sup></b> <b>Q1 (ref):</b> 65.55 <b>Q2:</b> 83.58 <b>Q3:</b> 96.44 <b>Q4:</b> 110.51 <b>Q5:</b> 134.2  <b>Person years:</b> <b>Q1 (ref):</b> 44,414 <b>Q2:</b> 44,580 <b>Q3:</b> 44,464 <b>Q4:</b> 44,607 <b>Q5:</b> 44,457	<b>Q1:</b> 215 <b>Q2:</b> 190 <b>Q3:</b> 183 <b>Q4:</b> 167 <b>Q5:</b> 163	<b>Model 1:</b> age, smoking status  <b>Model 2:</b> model 1 + BMI, frequency of vigorous exercise, alcohol consumption, postmenopausal hormone use, multivitamin use, history of hypertension, history of elevated cholesterol, parental history of T2DM.	<b>Model 1; RR (95% CI)</b> <b>Q1:</b> 1 <b>Q2:</b> 0.87 (0.72-0.84) <b>Q3:</b> 0.84 (0.68-1.02) <b>Q4:</b> 0.75 (0.61-0.92) <b>Q5:</b> 0.73 (0.59-0.89) <b>P per trend = 0.0007</b>  <b>Model 2; RR (95% CI)</b> <b>Q1:</b> 1 <b>Q2:</b> 0.94 (0.77-1.15) <b>Q3:</b> 0.88 (0.72-1.08) <b>Q4:</b> 0.92 (0.74-1.14) <b>Q5:</b> 0.86 (0.69-1.06) <b>P per trend = 0.17</b>

<sup>20</sup> WHO. Diabetes mellitus: report of a WHO study group. Geneva: WHO; 1985.

<sup>21</sup> American Diabetes Association: Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care* 26:5S–20S, 2003

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		<b>Sex:</b> Females <b>Ethnicity:</b> 94.8 White, 2.3% African American, 1.1% Hispanic, 1.4% Asian/Pacific Islander, 0.3% American Indian/Alaskan Native, and 0.1% more than one race. <b>Age:</b> ≥45 y	compared with medical records in a validation study.	<b>Exposure assessment:</b> SFFQ			
2	<b>EPIC-InterAct</b>  8 European countries  Sluijs et al. (2013)  12 y (median)  <b>Prospective case-cohort</b>  Public funding	<b>N</b> = 27,779  <b>Population sampled:</b> mainly general population recruited in 26 centres  <b>Excluded:</b> prevalent diabetes, unknown diabetes status, abnormal energy intake (top 1% and bottom 1%, or over-estimated energy requirement), missing information on nutritional intake or other covariates.  <b>n</b> = 26,088  <b>Random sub-cohort</b> n = 16,835 <b>Incident T2DM cases</b> n = 12,403 <b>Sex:</b> 62% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 35-70 y	Incident cases identified through self-report, linkage to primary and secondary care registers, medication use, hospital admissions and mortality data. Identified cases were verified with further evidence, including individual medical record reviews.  Ascertainment of self-reported cases and identification of new cases through other sources varied from country to country <sup>22</sup>  Diagnostic criteria for incident diabetes NR	<b>g/d (median)<sup>+</sup></b> <u>Q1 (ref):</u> 65 <u>Q2:</u> 88 <u>Q3:</u> 108 <u>Q4:</u> 137  <b>n</b> <u>Q1 (ref):</u> 3,815 <u>Q2:</u> 3,814 <u>Q3:</u> 3,815 <u>Q4:</u> 3,814  <b>Exposure assessment:</b> Quantitative dietary questionnaire or SFFQ (country dependent)	<u>Q1 (ref):</u> 3,251 <u>Q2:</u> 2,872 <u>Q3:</u> 2,741 <u>Q4:</u> 2,695	<b>Model 1:</b> age, sex, centre  <b>Model 2:</b> model 1 + education, physical activity, BMI, menopausal status, smoking status, alcohol consumption  <b>Model 3:</b> model 2 + energy intake, dietary protein, polyunsaturated: saturated fat ratio and fibre	<b>Model 1; HR (95% CI)</b> <u>Q1:</u> 1 <u>Q2:</u> 0.86 (0.76, 0.96) <u>Q3:</u> 0.81 (0.71, 0.92) <u>Q4:</u> 0.76 (0.62, 0.93) <b>P per tend = 0.01</b>  <b>Model 2; HR (95% CI)</b> <u>Q1:</u> 1 <u>Q2:</u> 0.95 (0.84, 1.08) <u>Q3:</u> 0.86 (0.78, 0.94) <u>Q4:</u> 0.90 (0.80, 1.03) <b>P per tend=0.04</b>  <b>Model 3; HR (95% CI)</b> <u>Q1:</u> 1 <u>Q2:</u> 0.98 (0.86, 1.11) <u>Q3:</u> 0.89 (0.81, 0.99) <u>Q4:</u> 0.96 (0.86, 1.07) <b>P per tend=0.31</b>

<sup>22</sup> InterAct consortium. Design and cohort description of the InterAct Project: an examination of the interaction of genetic and lifestyle factors on the incidence of type 2 diabetes in the EPIC Study. Diabetologia. 2011;54:2272–82

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
2	WHI  USA  Tasevska et al. (2018)  Up to 16 y  Public funding	N = 122,970  <b>Population sampled:</b> Postmenopausal women recruited from 40 clinical centres  <b>Excluded:</b> implausible self-reported energy intake (<600 or >5000 kcal/day) on the FFQ or missing data on relevant covariates, prevalent cases of T2DM at baseline.  <b>Follow-up rate:</b> 99.5%  n = 75,320  <b>Sex:</b> Females <b>Ethnicity:</b> ~ 84% Caucasian, 7.6% Black, Hispanic/Latino 4% and 3% Asian/Pacific <b>Age:</b> 50-79 y	Self-reported incident cases identified via annual mailed questionnaires. Participants asked about having been prescribed pills or insulin for diabetes.  Positive predictive value of incident T2DM = 82.2%.  Negative predictive value when diabetes is not reported = 94.5%.  as compared with medical records in a validation study <sup>23</sup> , according to the ADA criteria (1997) <sup>24</sup> .	<b>Geometric mean (95%CI)</b>  *Uncalibrated (g/day): 94 (69, 124)  Uncalibrated density (g/1000 kcal): 61.9 (61.8, 62.0)  *Calibrated (g/d): 189 (155, 228)  Calibrated <sup>25</sup> density (g/1000 kcal): 84.3 (84.1, 84.6)  n = 75,320  <b>Exposure assessment:</b> SFFQ	6,621	<u>Model 1:</u> age, energy intake (total energy intake in <b>energy substitution</b> models; non-sugars and non-alcohol energy in <b>energy partition</b> models)  <u>Model 2:</u> model 1 + race and ethnicity, marital status, education, smoking status, postmenopausal hormone therapy use, history of treated hypertension or hypercholesterolemia, history of CVD, family history of T2DM, alcohol consumption, activity-related energy expenditure  <u>Model 3:</u> model 2 + BMI and WC	<b>HR (95% CI) for a 20%<sup>26</sup> increase in:</b>  <b>Uncalibrated TS intake ES models:</b> <u>Model 1:</u> 0.93 (0.92, 0.95) <u>Model 2:</u> 0.92 (0.90, 0.95) <u>Model 3:</u> 0.95 (0.94, 0.97)  <b>EP models:</b> <u>Model 1:</u> 0.94 (0.93, 0.96) <u>Model 2:</u> 0.94 (0.93, 0.95) <u>Model 3:</u> 0.96 (0.95, 0.98)  <b>Calibrated TS intake ES models:</b> <u>Model 1:</u> 0.99 (0.92, 1.07) <u>Model 2:</u> 0.94 (0.76, 1.15) <u>Model 3:</u> 0.93 (0.67, 1.31)  <b>EP models:</b> <u>Model 1:</u> 1.22 (1.09, 1.37) <u>Model 2:</u> 1.00 (0.85, 1.18) <u>Model 3:</u> 0.94 (0.87, 1.01)
<b>Exposure: added sugars</b>							
2	MDCS  Sweden  Sonestedt et al. (2012)*	N = 28,098  <b>Population sampled:</b> general population from the city of Malmö  <b>Excluded:</b> cases of diabetes at baseline,	Identified via the Swedish National Diabetes Register, the Diabetes 2000 register of Scania (both require physician diagnosis against established criteria), and the Malmö HbA1c registry (two values >6.9% needed for diagnosis)	<b>Non-alcohol E% (range)</b> <u>Q1 (ref):</u> 0.0-6.6 <u>Q2:</u> 6.6-8.6 <u>Q3:</u> 8.6-10.6 <u>Q4:</u> 10.6-13.3 <u>Q5:</u> 13.3-56.1	<u>Q1:</u> 890 <u>Q2:</u> 794 <u>Q3:</u> 805 <u>Q4:</u> 787 <u>Q5:</u> 770	<b>Model 1:</b> sex, age, diet-method version, season, and total energy intake  <b>Model 2:</b> model 1 + physical activity, alcohol intake, smoking, and education	<b>Model 1; HR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.87 (0.79, 0.96) <u>Q3:</u> 0.87 (0.79, 0.96) <u>Q4:</u> 0.87 (0.79, 0.95) <u>Q5:</u> 0.86 (0.78, 0.94) <b>P per trend = 0.004</b>

<sup>23</sup> Jackson JM, DeFor TA, Crain AL, et al. Validity of diabetes self-reports in the Women's Health Initiative. Menopause. 2014;21(8):861-868

<sup>24</sup> American Diabetes Association Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetes Care. 1997 Jul;20:1183-97

<sup>25</sup> Calibration equations were derived for TS, energy, protein, NA/K intake ratio, and activity-related energy expenditure

<sup>26</sup> Corresponding to 18.0 g/1,000 kcal for calibrated and 12.6 g/1,000 kcal for uncalibrated TS

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	18.4 y (mean)  Public funding	missing data on physical activity, tobacco, or alcohol  n = 26,622  Sex: 61% females Ethnicity: Caucasian Age: 45-73 y		n/person-years Q1 (ref): 5306/96712 Q2: 5322/98432 Q3: 5329/99684 Q4: 5338/98246 Q5: 5327/96111  Exposure assessment: 7-d food record and SFFQ		Model 3: model 2 + BMI  Model 4: model 3 + coffee, meat, whole grains, soft drinks	Model 2; HR (95%CI) Q1 (ref): 1 Q2: 0.88 (0.80, 0.97) Q3: 0.87 (0.79, 0.96) Q4: 0.85 (0.77, 0.94) Q5: 0.80 (0.72, 0.88) P per trend < 0.001  Model 3; HR (95%CI) Q1 (ref): 1 Q2: 0.94 (0.85, 1.03) Q3: 0.96 (0.87, 1.06) Q4: 0.97 (0.88, 1.07) Q5: 0.94 (0.85, 1.04) P per trend = 0.451  Model 4; HR (95%CI) Q1 (ref): 1 Q2: 0.94 (0.86, 1.04) Q3: 0.97 (0.88, 1.07) Q4: 0.98 (0.88, 1.08) Q5: 0.96 (0.86, 1.07) P per trend = 0.685
Exposure: sucrose							
1	EPIC-Norfolk  UK  Ahmadi-Abhari et al. (2014)  10 y  Prospective case-cohort	Same population and exclusion criteria as for total sugars	Same ascertainment of the outcome as for total sugars	g/d Mean (SD) 49.3±27.0  E% Mean (SD) 9.3±4.0  E% (median) Q1 (ref): 5 Q2: 7.5 Q3: 9.3 Q4: 11.4 Q5: 15.3  n	Q (E%) Q1 (ref): 184 Q2: 147 Q3: 124 Q4: 144 Q5: 154	Model 1: age, sex  Model 2: model 1 + total energy intake, family history of T2DM, smoking, alcohol intake, physical activity, level of education, BMI	HR (95%CI) per each SD (27g) M 1: 0.89 (0.81, 0.97) M 2: 1.00 (0.88, 1.12)  Model 1; HR (95%CI) by Q (E%) Q1 (ref): 1 Q2: 0.77 (0.61, 0.99) Q3: 0.68 (0.53, 0.88) Q4: 0.71 (0.56, 0.92) Q5: 0.71 (0.56, 0.92)  Model 2; HR (95%CI) by Q (E%) Q1 (ref): 1



RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Public funding			Q1 (ref): 846 Q2: 824 Q3: 793 Q4: 846 Q5: 844  Exposure assessment: 7-d food diary			Q2: 0.87 (0.64, 1.18) Q3: 0.84 (0.62, 1.14) Q4: 0.98 (0.72, 1.33) Q5: 0.91 (0.69, 1.23)
1	FMCHES  Finland  Montonen et al. (2007)  12 y  Public funding	Same population and exclusion criteria as for total sugars	Same ascertainment of the outcome as for total sugars	g/d (median) <sup>†</sup> Q1 (ref): 28.5 Q2: 43.2 Q3: 56.7 Q4: 79.5  n Q1 (ref): 1,065 Q2: 1,071 Q3: 1,074 Q4: 1,074  Exposure assessment: DHI (including SFFQ)	Q1 (ref): 42 Q2: 43 Q3: 51 Q4: 39	Model 1: age, sex, BMI, energy intake, smoking, geographic area, physical activity, family history of diabetes  Model 2: model 1 + prudent dietary pattern score, conservative pattern score  Model 3: model 2 + serum cholesterol, blood pressure, history of infarction, history of angina pectoris and history of cardiac failure  <i>Adjustments as specified in Model 2 did not materially change the RRs as estimated in Model 1 (not shown)</i>	Model 1; RR (95% CI) Q1 (ref): 1 Q2: 1.21 (0.79, 1.87) Q3: 1.33 (0.88, 2.02) Q4: 1.12 (0.71, 1.76) P per trend=0.60  Model 3; RR (95% CI) Q1 (ref): 1 Q2: 1.25 (0.81, 1.94) Q3: 1.48 (0.97, 2.25) Q4: 1.22 (0.77, 1.92) P per trend=0.35
1	WHS  USA  Janket et al. (2003)*  6 y (median)  Public funding	Same population and exclusion criteria as for total sugars	Same ascertainment of the outcome as for total sugars	g/d (median) <sup>†</sup> Q1 (ref): 25.8 Q2: 33.6 Q3: 39.3 Q4: 45.8 Q5: 57.2  Person years: Q1 (ref): 44,362 Q2: 44,298 Q3: 44,549 Q4: 44,567 Q5: 44,746	Q1: 196 Q2: 194 Q3: 175 Q4: 188 Q5: 165	Model 1: age, smoking status  Model 2: model 1 + BMI, frequency of vigorous exercise, alcohol consumption, postmenopausal hormone use, multivitamin use, history of hypertension, history of elevated cholesterol, parental history of T2DM.	Model 1; RR (95% CI) Q1 (ref): 1 Q2: 0.99 (0.81, 1.21) Q3: 0.89 (0.72, 1.09) Q4: 0.95 (0.77, 1.16) Q5: 0.82 (0.66, 1.01) P per trend = 0.06  Model 2; RR (95% CI) Q1 (ref): 1 Q2: 1.00 (0.81, 1.23) Q3: 0.98 (0.79, 1.22) Q4: 1.00 (0.81, 1.24)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
				Exposure assessment: SFFQ			Q5: 0.84 (0.67, 1.04) P per trend = 0.16
2	MDCS  Sweden  Sonestedt et al. (2012) *  18.4 y (mean)  Public funding	Same population and exclusion criteria as for total added sugars	Same ascertainment of the outcome as for total added sugars	E% (range) Q1 (ref): 0.5-5.8 Q2: 5.8-7.4 Q3: 7.4-9.0 Q4: 9.0-11.1 Q5: 11.1-38.6  n/person-years Q1 (ref): 5300/95507 Q2: 5333/99975 Q3: 5335/98759 Q4: 5331/99145 Q5: 5323/95799  Exposure assessment: 7-d food record and SFFQ	Q1: 894 Q2: 761 Q3: 841 Q4: 756 Q5: 794	Model 1: sex, age, diet-method version, season, and total energy intake  Model 2: model 1 + physical activity, alcohol intake, smoking, and education  Model 3: model 2 + BMI  Model 4: model 3 + coffee, meat, whole grains, soft drinks	Model 1; HR (95%CI) Q1 (ref): 1 Q2: 0.86 (0.79, 0.97) Q3: 0.98 (0.89, 1.08) Q4: 0.89 (0.80, 0.98) Q5: 0.97 (0.88, 1.06) P per trend = 0.687  Model 2; HR (95%CI) Q1 (ref): 1 Q2: 0.88 (0.78, 0.95) Q3: 1.00 (0.91, 1.10) Q4: 0.88 (0.80, 0.97) Q5: 0.90 (0.82, 1.00) P per trend = 0.083  Model 3; HR (95%CI) Q1 (ref): 1 Q2: 0.91 (0.83, 1.00) Q3: 1.06 (0.96, 1.17) Q4: 0.96 (0.87, 1.06) Q5: 1.00 (0.91, 1.11) P per trend = 0.646  Model 4; HR (95%CI) Q1 (ref): 1 Q2: 0.91 (0.83, 1.01) Q3: 1.07 (0.97, 1.18) Q4: 0.97 (0.87, 1.07) Q5: 1.03 (0.92, 1.15) P per trend = 0.404
Exposure: free glucose							
1	EPIC-Norfolk  UK	Same population and exclusion criteria as for total sugars	Same ascertainment of the outcome as for total sugars	g/d Mean (SD) 17.1±8.4	Q1 (ref): 200 Q2: 161 Q3: 138 Q4: 132	Model 1: age, sex  Model 2: model 1+ total energy intake, family history of T2DM,	HR (95%CI) per each SD (8g/d) M 1: 0.83 (0.75, 0.90) M 2: 0.91 (0.82, 1.02)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Ahmadi-Abhari et al. (2014)  10 y  <b>Prospective case-cohort</b>  Public funding			<b>E%</b> <b>Mean (SD)</b> 3.3±1.5  <b>E% (median)</b> <u>Q1 (ref):</u> 1.6 <u>Q2:</u> 2.6 <u>Q3:</u> 3.4 <u>Q4:</u> 4.2 <u>Q5:</u> 5.6  <b>n</b> <u>Q1 (ref):</u> 862 <u>Q2:</u> 848 <u>Q3:</u> 831 <u>Q4:</u> 818 <u>Q5:</u> 794  <b>Exposure assessment:</b> 7-d food diary	<u>Q5:</u> 122	smoking, alcohol intake, physical activity, level of education, BMI	<b>Model 1; HR (95%CI) by Q (E%)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.82 (0.65, 1.03) <u>Q3:</u> 0.67 (0.53, 0.86) <u>Q4:</u> 0.65 (0.51, 0.84) <u>Q5:</u> 0.63 (0.50, 0.82)  <b>Model 2; HR (95%CI) by Q (E%)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.84 (0.65, 1.11) <u>Q3:</u> 0.72 (0.55, 0.94) <u>Q4:</u> 0.74 (0.56, 0.99) <u>Q5:</u> 0.82 (0.62, 1.11)
<b>1</b>	<b>FMCHES</b>  Finland  Montonen et al. (2007)  12 y  Public funding	Same population and exclusion criteria as for total sugars	Same ascertainment of the outcome as for total sugars	<b>g/d (median)†</b> <u>Q1 (ref):</u> 5.6 <u>Q2:</u> 10.6 <u>Q3:</u> 15.9 <u>Q4:</u> 27.5  <b>n</b> <u>Q1 (ref):</u> 1,074 <u>Q2:</u> 1,068 <u>Q3:</u> 1,069 <u>Q4:</u> 1,073  <b>Exposure assessment:</b> DHI (including SFFQ)	<u>Q1 (ref):</u> 41 <u>Q2:</u> 38 <u>Q3:</u> 37 <u>Q4:</u> 59	<b>Model 1:</b> age, sex, BMI, energy intake, smoking, geographic area, physical activity, family history of diabetes  <b>Model 2:</b> model 1 + prudent dietary pattern score, conservative pattern score  <b>Model 3:</b> model 2 + serum cholesterol, blood pressure, history of infarction, history of angina pectoris and history of cardiac failure  <b>Adjustments as specified in Model 2 did not materially change the RRs as estimated in Model 3 (not shown)</b>	<b>Model 1; RR (95% CI)</b> <u>Q1:</u> 1 <u>Q2:</u> 0.96 (0.62, 1.50) <u>Q3:</u> 0.97 (0.62, 1.53) <u>Q4:</u> 1.57 (1.04, 2.37) <b>P per trend = 0.01</b>  <b>Model 3; RR (95% CI)</b> <u>Q1:</u> 1 <u>Q2:</u> 0.98 (0.62, 1.55) <u>Q3:</u> 1.08 (0.68, 1.72) <u>Q4:</u> 1.68 (1.06, 2.65) <b>P per trend = 0.009</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
1	WHS  USA  Janket et al. (2003)*  6 y (median)  Public funding	Same population and exclusion criteria as for total sugars	Same ascertainment of the outcome as for total sugars	g/d (median) <sup>†</sup> Q1 (ref): 10.89 Q2: 15.21 Q3: 18.96 Q4: 23.27 Q5: 31.17  Person years: Q1 (ref): 44,693 Q2: 44,426 Q3: 44,470 Q4: 44,626 Q5: 44,308  Exposure assessment: SFFQ	Q1: 203 Q2: 192 Q3: 178 Q4: 168 Q5: 177	Model 1: age, smoking status  Model 2: model 1 + BMI, frequency of vigorous exercise, alcohol consumption, postmenopausal hormone use, multivitamin use, history of hypertension, history of elevated cholesterol, parental history of T2DM.	Model 1; RR (95% CI) Q1: 1 Q2: 0.95 (0.78, 1.16) Q3: 0.87 (0.71, 1.06) Q4: 0.81 (0.66, 0.99) Q5: 0.85 (0.70, 1.05) P per trend = 0.04  Model 2; RR (95% CI) Q1: 1 Q2: 1.08 (0.88, 1.33) Q3: 1.02 (0.82, 1.26) Q4: 0.96 (0.77, 1.19) Q5: 1.04 (0.85, 1.28) P per trend = 0.91
Exposure: free fructose							
1	EPIC-Norfolk  UK  Ahmadi-Abhari et al. (2014)  10 y  Prospective case-cohort  Public funding	Same population and exclusion criteria as for total sugars	Same ascertainment of the outcome as for total sugars	g/d Mean (SD) 18.4±9.6  E% Mean (SD) 3.6±1.9  E% (median) Q1 (ref): 1.6 Q2: 2.7 Q3: 3.6 Q4: 4.6 Q5: 6.4  n Q1 (ref): 880 Q2: 830 Q3: 826 Q4: 831 Q5: 786	Q (E%) Q1 (ref): 207 Q2: 147 Q3: 138 Q4: 146 Q5: 115	Model 1: age, sex  Model 2: model 1+ total energy intake, family history of T2DM, smoking, alcohol intake, physical activity, level of education, BMI	HR (95%CI) per each SD (10g/d) M 1: 0.82 (0.75, 0.91) M 2: 0.88 (0.78, 0.99)  Model 1; HR (95%CI) Q1 (ref): 1 Q2: 0.70 (0.55, 0.89) Q3: 0.66 (0.52, 0.84) Q4: 0.69 (0.54, 0.88) Q5: 0.60 (0.47, 0.79)  Model 2; HR (95%CI) Q1 (ref): 1 Q2: 0.75 (0.58, 0.98) Q3: 0.68 (0.52, 0.91) Q4: 0.76 (0.56, 1.00) Q5: 0.65 (0.47, 0.88)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
1	FMCHES Finland Montonen et al. (2007) up to 12 y Public funding	Same population and exclusion criteria as for total sugars	Same ascertainment of the outcome as for total sugars	Exposure assessment: 7-d food diary  g/d (median) <sup>†</sup> Q1 (ref): 6.0 Q2: 11.3 Q3: 17.0 Q4: 28.8  n Q1 (ref): 1,073 Q2: 1,070 Q3: 1,068 Q4: 1,073  Exposure assessment: DHI (including SFFQ)	Q1 (ref): 40 Q2: 41 Q3: 39 Q4: 55	Model 1: age, sex, BMI, energy intake, smoking, geographic area, physical activity, family history of diabetes  Model 2: model 1 + prudent dietary pattern score, conservative pattern score  Model 3: model 2 + serum cholesterol, blood pressure, history of infarction, history of angina pectoris and history of cardiac failure  <i>Adjustments as specified in Model 2 did not materially change the RRs as estimated in Model 3 (not shown)</i>	Model 1; RR (95% CI) Q1: 1 Q2: 1.08 (0.69, 1.67) Q3: 1.11 (0.71, 1.75) Q4: 1.52 (1.00, 2.32) P per trend = 0.03  Model 3; RR (95% CI) Q1: 1 Q2: 1.12 (0.71, 1.76) Q3: 1.22 (0.76, 1.96) Q4: 1.62 (1.01, 2.59) P per trend = 0.03
1	WHS USA Janket et al. (2003)* 6 y (median) Public funding	Same population and exclusion criteria as for total sugars	Same ascertainment of the outcome as for total sugars	g/d (median) <sup>†</sup> Q1 (ref): 11.19 Q2: 16.38 Q3: 20.63 Q4: 25.38 Q5: 34.28  Person years: Q1 (ref): 44,564 Q2: 44,515 Q3: 44,479 Q4: 44,587 Q5: 44,379  Exposure assessment: SFFQ	Q1: 208 Q2: 189 Q3: 175 Q4: 177 Q5: 169	Model 1: age, smoking status  Model 2: model 1 + BMI, frequency of vigorous exercise, alcohol consumption, postmenopausal hormone use, multivitamin use, history of hypertension, history of elevated cholesterol, parental history of type 2 diabetes.	Model 1; RR (95% CI) Q1: 1 Q2: 0.90 (0.74, 1.10) Q3: 0.83 (0.68, 1.02) Q4: 0.83 (0.68, 1.02) Q5: 0.79 (0.65, 0.97) P per trend = 0.02  Model 2; RR (95% CI) Q1: 1 Q2: 0.99 (0.81, 1.22) Q3: 1.04 (0.85, 1.29) Q4: 1.03 (0.83, 1.27) Q5: 0.96 (0.78, 1.19) P per trend = 0.86
Exposure: SSSD							

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
1	<b>BWHS</b>  USA  Palmer et al. (2008)  10 y  Public funding	<b>N</b> = 59,000  <b>Population sampled:</b> African American women from all regions of USA  <b>Excluded:</b> reported diabetes, GDM, myocardial infarction, stroke or cancer at baseline; pregnant at baseline; <30 years at the end of follow-up; data on height or weight missing at baseline; dietary questionnaire not completed or more than 10 dietary questions blank; implausible energy intake values (<500 or >3800 kcal); missing data on soft drink consumption in 1995  <b>n</b> = 43,960 <b>Sex:</b> females <b>Ethnicity:</b> African American <b>Age:</b> 21-69 y	Self-reported incident cases identified via bi-annual mailed questionnaires  Average response rate = 80%  Positive predictive value of incident diabetes = 94% as compared with medical records in a validation study including 293 women self-reporting new diagnosis of T2DM  Negative predictive value when diabetes is not reported = NR  Criteria to ascertain cases = NR	<b>Servings/time (range)</b> <b>C1 (ref):</b> <1/mo <b>C2:</b> 1-7/mo <b>C3:</b> 2-6/wk <b>C4:</b> 1/d <b>C5:</b> ≥2/d  Serving size = 12 oz (336 g)  <b>Person-years</b> <b>C1 (ref):</b> 96,266 <b>C2:</b> 111,418 <b>C3:</b> 78,319 <b>C4:</b> 29,273 <b>C5:</b> 23,608  <b>Exposure assessment:</b> SFFQ	<b>C1 (ref):</b> 733 <b>C2:</b> 783 <b>C3:</b> 656 <b>C4:</b> 280 <b>C5:</b> 261	<b>Model 1:</b> age (only IRR and not 95%CI given for this model)  <b>Model 2:</b> model 1 + family history of diabetes, physical activity, cigarette smoking, years of education and each of the 2 other types of drinks (SSFD/FJ and 100% FJ)  <b>Model 3:</b> model 2 + intake of red meat, processed meat, cereal fibre, coffee and GI  <b>Model 4:</b> model 3 + BMI (only IRR and 95%CI for C5 are reported in the paper)  <b>Model 5:</b> model 4 + energy intake  <b>Authors report that adjustments as specified in Model 5 did not materially change the RRs as estimated in Model 4 (data for model 5 are not reported in the paper)</b>	<b>Model 1; IRR</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.01 <b>C3:</b> 1.24 <b>C4:</b> 1.43 <b>C5:</b> 1.76  <b>Model 2; IRR (95%CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 0.96 (0.87, 1.06) <b>C3:</b> 1.14 (1.02, 1.27) <b>C4:</b> 1.27 (1.12, 1.47) <b>C5:</b> 1.51 (1.31, 1.75)  <b>Model 3; IRR (95%CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 0.89 (0.80, 0.99) <b>C3:</b> 1.00 (0.89, 1.12) <b>C4:</b> 1.11 (0.96, 1.28) <b>C5:</b> 1.24 (1.06, 1.45) <b>P per trend = 0.002</b>  <b>Model 4; IRR (95%CI)</b> <b>C5:</b> 1.05 (0.90, 1.23)
2	<b>FMCHES</b>  Finland  Montonen et al. (2007)  12 y	<b>Same and exclusion criteria as for total sugars + no data for SSSD consumption</b>  <b>n</b> = 2,360  <b>Sex:</b> 47% females <b>Ethnicity:</b> Caucasian	<b>Same ascertainment of the outcome as for total sugars</b>	<b>g/d (median)</b> <b>Q1 (ref):</b> 0 <b>Q2:</b> 1 <b>Q3:</b> 13 <b>Q4:</b> 143  <b>n</b> <b>Q1 (ref):</b> 741 <b>Q2:</b> 458	<b>Q1 (ref):</b> 25 <b>Q2:</b> 12 <b>Q3:</b> 21 <b>Q4:</b> 33	<b>Model 1:</b> age, sex, BMI, energy intake, smoking, geographic area, physical activity, family history of diabetes  <b>Model 2:</b> model 1 + prudent dietary pattern score, conservative pattern score	<b>Model 1; RR (95% CI)</b> <b>Q1:</b> 1 <b>Q2:</b> 0.78 (0.39, 1.58) <b>Q3:</b> 0.97 (0.54, 1.76) <b>Q4:</b> 1.61 (0.94, 2.74) <b>P per trend=0.02</b>  <b>Model 3; RR (95% CI)</b> <b>Q1:</b> 1

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	Public funding	Age: 40-69 y		Q3: 573 Q4: 588  Exposure assessment: DHI (including SFFQ)		<b>Model 3:</b> model 2 + serum cholesterol, blood pressure, history of infarction, history of angina pectoris and history of cardiac failure  <i>Adjustments as specified in Model 2 did not materially change the RRs as estimated in Models 1 or 3 (not shown)</i>	Q2: 0.85 (0.42, 1.73) Q3: 0.80 (0.43, 1.49) Q4: 1.60 (0.93, 2.76) <b>P per trend=0.01</b>
<b>3</b>	<b>KoGES</b>  South Korea  Kang and Kim (2017)  5.7 y (mean)  Public funding	<b>N=</b> 10,030  <b>Population sampled:</b> general population living in Ansan (urban) and Ansong (rural) areas  <b>Excluded:</b> participants who refused to participate in follow-up examinations, insufficient information, non-responders to dietary examination and prevalence of CVD or cancer  <b>n</b> = 6,660 Females: 3,592 Males: 3,068  <b>Follow-up rate:</b> 63.3 %  <b>Ethnicity:</b> Asian  <b>Age:</b> 40-69 y	The blood samples were collected after at least 8 h of fasting at baseline and during every follow-up examination.  Incident <b>high fasting blood glucose</b> defined as FBG $\geq$ 5.6 mmol/l, current use of insulin or oral hypoglycaemic medication, diabetes diagnosis by a physician.	<b>Servings/week (range)</b> <b>C1:</b> Rarely or never <b>C2:</b> $<1$ <b>C3:</b> $\geq 1$ to $<4$ <b>C4:</b> $\geq 4$  <b>n</b> females <b>C1:</b> 1,809 <b>C2:</b> 1,319 <b>C3:</b> 407 <b>C4:</b> 57  males: <b>C1:</b> 1,042 <b>C2:</b> 1,223 <b>C3:</b> 678 <b>C4:</b> 125  <b>Serving size:</b> 200 ml  <b>Exposure assessment:</b> SFF Q	<b>Females:</b> <b>C1:</b> 458 <b>C2:</b> 317 <b>C3:</b> 120 <b>C4:</b> 16  <b>Males:</b> <b>C1:</b> 416 <b>C2:</b> 443 <b>C3:</b> 264 <b>C4:</b> 58	<b>Model 1:</b> age  <b>Model 2:</b> age, income level, education level, alcohol consumption, smoking status, physical activity, BMI, energy intake, percentage of fat, fibre intake and the presence of diseases	<b>Females</b>  <b>Model 1; HR (95% CI)</b> <b>C1</b> (ref): 1 <b>C2:</b> 0.93 (0.80, 1.07) <b>C3:</b> 1.33 (1.08, 1.62) <b>C4:</b> 1.37 (0.83, 2.26) <b>P per trend=0.058</b>  <b>Model 2; HR (95% CI)</b> <b>C1</b> (ref): 1 <b>C2:</b> 0.90 (0.78, 1.04) <b>C3:</b> 1.23 (1.00, 1.51) <b>C4:</b> 1.13 (0.68, 1.86) <b>P per trend=0.36</b>  <b>Males</b>  <b>Model 1; HR (95% CI)</b> <b>C1</b> (ref): 1 <b>C2:</b> 0.80 (0.70, 0.91) <b>C3:</b> 0.97 (0.83, 1.14) <b>C4:</b> 1.20 (0.91, 1.59) <b>P per trend=0.77</b>  <b>Model 2; HR (95% CI)</b> <b>C1</b> (ref): 1 <b>C2:</b> 0.80 (0.70, 0.92) <b>C3:</b> 0.97 (0.82, 1.13) <b>C4:</b> 1.12 (0.85, 1.49) <b>P per trend=0.95</b>



RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: SSSD + SSFD</b>							
<b>1</b>	<b>Framingham-Offspring</b>  USA  Ma et al. (2016a)  14 y (median)  Public funding	<b>N</b> = 5,135  <b>Population sampled:</b> offspring of Original Cohort (sampled from the general population of Framingham) and their spouses  <b>Excluded:</b> not report of beverage exposure, prediabetes or T2DM at baseline, missing prediabetes status at baseline or follow-up, missing data on covariates.  <b>n</b> = 1,751  <b>Sex:</b> 59.6 % females <b>Ethnicity:</b> Caucasian <b>Age:</b> 30-59 y	<b>Composite outcome:</b> incidence of prediabetes or T2DM  <b>At baseline: T2DM</b> defined as an FPG $\geq 7$ mmol/L, a 2-h OGTT glucose concentration $\geq 11.1$ mmol/L, or the reported use of hypoglycemic medications; <b>prediabetes</b> defined as an FPG $\geq 5.6$ and $< 7$ mmol/L or a 2-h OGTT glucose concentration $\geq 7.8$ and $< 11.1$ mmol/L without the use of hypoglycemic medications (FPG and OGTT performed at baseline)  <b>At follow-up: incident T2DM</b> defined as first occurrence of FPG $\geq 7$ mmol/L or use of hypoglycemic medications; <b>incident prediabetes</b> defined as first occurrence of an FPG $\geq 5.6$ and $< 7$ mmol/L in absence of hypoglycemic medications (only FPG measured at follow-up).	<b>servings/wk (median)</b>  <u>Q1 (ref):</u> 0 <u>Q2:</u> 0.5 <u>Q3:</u> 2 <u>Q4:</u> 6  Serving size = 12 fl oz (360 mL)  <b>n</b> <u>Q1 (ref):</u> 403 <u>Q2:</u> 475 <u>Q3:</u> 435 <u>Q4:</u> 438  <b>Exposure assessment:</b> SFFQ  <i>Cumulative intake (i.e. mean intake reported at examinations up to and including the examination of prediabetes diagnosis)</i>	<u>Q1 (ref):</u> 191 <u>Q2:</u> 221 <u>Q3:</u> 207 <u>Q4:</u> 270	<b>Model 1:</b> age and sex  <b>Model 2:</b> model 1 + baseline fasting glucose, smoking, hypertension, physical activity, BMI, energy intake, alcohol intake, fruit juice intake, diet soda intake, Dietary Guidelines Adherence Index (DGA1) score  <b>Model 3:</b> model 2 + BMI change  <b>Model 4:</b> model 2 except DGA1 score was replaced with intake of individual foods including coffee, whole grains, vegetables, red meat, nuts, and fish.  <b>Model 5:</b> model 4 + BMI change  <b>Adjustments as specified in Models 4 and 5 did not materially change the RRs as estimated in Model 1; adjustments as specified in Model 3 did not materially change the RRs as estimated in Model 2 (not shown)</b>	<b>Cumulative intake</b>  <b>Model 1; HR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.95 (0.78, 1.15) <u>Q3:</u> 0.90 (0.73, 1.09) <u>Q4:</u> 1.29 (1.06, 1.57) <b>P per trend &lt; 0.001</b>  <b>Model 2; HR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.99 (0.81, 1.20) <u>Q3:</u> 0.95 (0.77, 1.17) <u>Q4:</u> 1.49 (1.20, 1.86) <b>P per trend &lt; 0.001</b>  <b>No association was observed for ASB</b> <b>Model 2; HR (95% CI)</b> <u>Q4 vs Q1:</u> 1.02 (0.85, 1.22) <b>P per trend = 0.22</b>
<b>1</b>	<b>HPFS</b>  USA  de Koning et al. (2011)  20 y	<b>N</b> = 51,529  <b>Population sampled:</b> male health professionals  <b>Excluded:</b> T1DM, T2DM, CVD (heart	Questionnaires were mailed every other year to participants to assess health status and lifestyle factors (94% response rate). Self-reported diagnoses of T2DM was verified with a supplementary questionnaire specific for T2DM. Cases before 1998 defined by National	<b>Servings/time Median (range)</b> <u>Q1 (ref):</u> never <u>Q2:</u> 2/mo <u>Q3:</u> 2/wk (1-4/wk) <u>Q4:</u> 6.5/wk (4.5/wk to 7.5/d)  <b>Servings/d (mean <math>\pm</math> SD)</b>	<u>Q1:</u> 586 <u>Q2:</u> 629 <u>Q3:</u> 685 <u>Q4:</u> 780	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + smoking, physical activity, alcohol intake, multivitamin use  <b>Model 3:</b> model 2 + family history of T2DM	<b>Model 1; HR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.00 (0.89, 1.13) <u>Q3:</u> 1.03 (0.92, 1.15) <u>Q4:</u> 1.25 (1.11, 1.39) <b>P per trend &lt; 0.01</b>  <b>Model 6; HR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.06 (0.94, 1.19)



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	Public funding	<p>attack, stroke, angina, or coronary artery bypass graft), cancer (except nonmelanoma skin cancer) or an implausible energy intake (&lt;800 or &gt;4200 kcal/d) at baseline</p> <p><b>n</b> = 40,389</p> <p><b>Sex:</b> Males <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 40–75 y</p>	<p>Diabetes Data Group criteria<sup>27</sup> and cases after 1998 defined by American Diabetes Association criteria (1997).</p> <p>Positive predictive value for incident T2DM = 97% as compared with medical records in a validation study<sup>28</sup></p> <p>Negative predictive value when diabetes is not reported = NR</p>	<p>0.36 ±0.61</p> <p>Serving size = 12oz (355mL)</p> <p><b>n/person-years</b> <b>Q1 (ref):</b> 13,675/167,462 <b>Q2:</b> 5,022/165,515 <b>Q3:</b> 11,729/189,851 <b>Q4:</b> 9,963/187,709</p> <p><b>Exposure assessment:</b> SFFQ</p>		<p><b>Model 4:</b> model 3 + high triglycerides at baseline, high blood pressure, and use of diuretics</p> <p><b>Model 5:</b> model 4 + previous weight change and being on a low-calorie diet</p> <p><b>Model 6:</b> model 5 + alternative Healthy Eating Index</p> <p><b>Model 7:</b> model 6 + total energy intake</p> <p><b>Model 8:</b> model 7 + BMI</p> <p><b>Adjustments as specified in Models 2, 3, 4 and 5 did not materially change the RRs as estimated in Model 1 (not shown)</b></p>	<p><b>Q3:</b> 1.05 (0.94, 1.18) <b>Q4:</b> 1.22 (1.09, 1.37) <b>P per trend=0.04</b></p> <p><b>Model 7; HR (95%CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 1.04 (0.92, 1.16) <b>Q3:</b> 1.01 (0.90, 1.13) <b>Q4:</b> 1.12 (0.99, 1.26) <b>P per trend=0.04</b></p> <p><b>Model 8; HR (95%CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 1.09 (0.97, 1.22) <b>Q3:</b> 1.07 (0.95, 1.20) <b>Q4:</b> 1.24 (1.09, 1.40) <b>P per trend &lt; 0.01</b></p> <p><b>HR (95%CI) per each serving</b> <b>M8:</b> 1.16 (1.08, 1.25)</p> <p><b>A positive (non-significant) association was observed for ASB</b> <b>Model 8; HR (95%CI)</b> <b>Q4 vs Q1:</b> 1.09 (0.98, 1.21) <b>P per trend = 0.13</b></p>
<b>2</b>	<b>CARDIA</b>  USA  Duffey et al. (2010)	<p><b>N</b> = 5,115</p> <p><b>Population sampled:</b> general population of 4 centres selected to balance subgroups of</p>	<p>Fasting glucose was obtained by venous blood draw. <b>High fasting glucose</b> was defined as ≥6.1 mmol/L or use of diabetic medication</p>	<p><b>Kcal/day (mean±SEM)</b></p> <p><b>Year 0:</b> n=5,034 167±3</p> <p><b>Year 7:</b> n= 3,877</p>	267	<p><b>Model:</b> race, gender, centre, age, weight, smoking status, energy from food, total physical activity, energy from other beverages (low-fat milk, whole-fat milk and fruit juice), and energy from alcohol.</p>	<p><b>Per 100 kcal increase* HR (95% CI)</b> 1.00 (0.94, 1.07)</p>

<sup>27</sup> National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. Diabetes 1979;28:1039-57

<sup>28</sup> Hu FB, Leitzmann MF, Stampfer MJ, Colditz GA, Willett WC, Rimm EB. Physical activity and television watching in relation to risk for type 2 diabetes mellitus in men. Arch Intern Med 2001;161:1542–8.

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	20 y  Mixed funding	<p>race, sex, education and age</p> <p><b>Excluded:</b> pregnancy, fasting &lt; 8 h at any examination (baseline, 7 and 20 y); high fasting plasma glucose or use of diabetic medication at baseline or 7-y visit</p> <p><b>n</b> = 2,160</p> <p><b>Sex:</b> 53.5 % females</p> <p><b>Ethnicity:</b> Caucasian 52.6%, Black 47.4%</p> <p><b>Age:</b> 18-30 y</p>		<p>196±8</p> <p>Average of intake at 0 and 7 years used for the analysis = NR</p> <p><b>Exposure assessment:</b> SFF Q</p>			
2	<p><b>EPIC-InterAct<sup>29</sup></b></p> <p>FR, UK, NL, DE, DK, SE</p> <p>InterAct consortium (2013)*</p> <p>16 y</p> <p><b>Prospective case-cohort</b></p> <p>Public funding</p>	<p><b>N</b> = 29,238</p> <p><b>Population sampled:</b> Mainly general population recruited from 6 EU countries</p> <p><b>Excluded:</b> diabetes at baseline, within the lowest and highest 1% of the cohort distribution of the ratio of reported total energy intake:energy requirement, with missing information on diet, physical</p>	<p>Ascertainment of incident T2DM involved a review of the existing EPIC datasets at each centre using multiple sources of evidence, including self-report, linkage to primary-care registers, secondary-care registers, medication use (drug registers), hospital admissions and mortality data.</p> <p>Information from any follow-up visit or external evidence with a date later than the baseline visit was used. Cases in Denmark and Sweden were not ascertained by self-report, but identified via local and national diabetes and pharmaceutical</p>	<p><b>Median, g/d (Servings/time, range)</b></p> <p><u>C1(ref):</u> 0(&lt;1/mo) <u>C2:</u> 19.3 (1-4/mo) <u>C3:</u> 94.3(&gt;1-6/wk) <u>C4:</u> 425.7 (≥1/d)</p> <p><b>Serving size</b> = 250 g</p> <p><b>n/category of intake:</b></p> <p><u>C1:</u> 9,150 <u>C2:</u> 2,187 <u>C3:</u> 3,531 <u>C4:</u> 1,137</p>	<p><u>C1(ref):</u> 3,948 <u>C2:</u> 964 <u>C3:</u> 1,599 <u>C4:</u> 605</p>	<p><b>Model 1:</b> crude</p> <p><b>Model 2:</b> sex, educational level, physical activity, smoking status and alcohol consumption, artificially sweetened soft drinks plus adjustment for juice consumption</p> <p><b>Model 3:</b> Model 2 + energy intake</p> <p><b>Model 4:</b> Model 3 + BMI</p>	<p><b>Model 1; HR (95% CI)</b> <u>C1(ref):</u> 1 <u>C2:</u> 1.14 (0.97, 1.35) <u>C3:</u> 1.16 (1.05, 1.28) <u>C4:</u> 1.68 (1.40, 2.02) <b>P for trend</b> = &lt;0.0001</p> <p><b>Model 2; HR (95% CI)</b> <u>C1(ref):</u> 1 <u>C2:</u> 1.13 (0.97, 1.31) <u>C3:</u> 1.04 (0.94, 1.15) <u>C4:</u> 1.39 (1.16, 1.67) <b>P for trend</b> = &lt;0.0001</p> <p><b>Model 3; HR (95% CI)</b> <u>C1(ref):</u> 1 <u>C2:</u> 1.12 (0.96, 1.31) <u>C3:</u> 1.04 (0.94, 1.15) <u>C4:</u> 1.39 (1.15, 1.69)</p>

<sup>29</sup> Data from individual countries was used for the dose-response meta-regression analysis as provided by the authors

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		activity, level of education, smoking status or BMI.  n = 16,164  <b>Random sub-cohort</b> n = 9,048 <b>Incident T2DM cases</b> n = 7,116  <b>Sex:</b> 62.5% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 35-70 y	registers and hence all ascertained cases were considered to be verified. To increase the specificity of the case definition for centres other than those from Denmark and Sweden, they sought further evidence for all cases with information on incident T2DM from fewer than two independent sources at a minimum, including individual medical records reviews in some centres.	<b>Exposure assessment:</b> SFF Q			<b>P for trend = 0.001</b>  <b>Model 4; HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 1.19 (0.91, 1.56) <u>C3</u> : 1.07 (0.94, 1.21) <u>C4</u> : 1.29 (1.02, 1.63) <b>P for trend = 0.013</b>  <b>HR (95% CI) per each 336 g increment</b> <u>M1</u> : 1.39 (1.21, 1.60) <u>M2</u> : 1.22 (1.09, 1.38) <u>M3</u> : 1.23 (1.08, 1.39) <u>M4</u> : 1.18 (1.06, 1.32)  <b>A positive non-significant association was observed for ASB</b> <b>Model 4; HR (95% CI)</b> <u>C4 vs C1</u> : 1.13 (0.85, 1.52) <b>P per trend = 0.24</b>  <b>HR (95% CI) per each 336 g increment of ASBs</b> <u>M4</u> : 1.11 (0.95, 1.31)
2	<b>NHS II</b>  USA  Schulze et al. (2004)  Up to 8 y  Public funding	<b>N</b> = 116,671  <b>Population sampled:</b> female nurses  <b>Excluded:</b> dietary questionnaire not completed in 1991 or if > 9 items were left blank; dietary intake reported was implausible	Women self-reporting new diagnosis of T2DM in the biennial questionnaire were sent supplementary questionnaires specific for T2DM. Diagnosis in accordance with the criteria of the National Diabetes Data Group <sup>7</sup> .  Positive predictive value for incident T2DM = 97-98% as	<b>servings/time (range)</b> <u>C1</u> (ref): <1/mo <u>C2</u> : 1-4/mo <u>C3</u> : 2-6/wk <u>C4</u> : ≥1/d  Serving size = 12 oz (355mL)  <b>n/person-years</b>	<u>C1</u> (ref): 368 <u>C2</u> : 163 <u>C3</u> : 95 <u>C4</u> : 115	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + alcohol intake, physical activity, family history of diabetes, smoking, post-menopausal hormone use, oral contraceptive use, intake of cereal fibre, magnesium, trans-fats and ratio of polyunsaturated to saturated fat, and consumption of ASSD and FJ	<b>Model 1; RR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 0.93 (0.78, 1.12) <u>C3</u> : 1.32 (1.06, 1.66) <u>C4</u> : 1.98 (1.60, 2.44) <b>P per trend &lt;0.001</b>  <b>Model 2; RR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 1.06 (0.87, 1.28) <u>C3</u> : 1.49 (1.16, 1.91)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		with regard to total energy intake (<500kcal/d or >3500kcal/d); history of diabetes, cancer or CVD at baseline; no data provided on physical activity in 1991.  <b>Follow-up rate</b> exceeding 90% for every 2-year period  <b>n</b> = 91,249  <b>Sex:</b> Females <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 24-44 y	compared with medical records in validation studies for the NSH and HPFS cohorts  Negative predictive value when diabetes is not reported = NR	<u>C1 (ref):</u> 49,203/381,275 <u>C2:</u> 23,398/188,501 <u>C3:</u> 9,950/ 80,086 <u>C4:</u> 8,698/ 66,438  <b>Exposure assessment:</b> SFFQ		<b>Model 3:</b> model 2 + BMI  <b>Model 4:</b> model 3 + total energy intake  <i><b>Only RR for the highest against the lowest intake categories are reported in the paper</b></i>	<u>C4:</u> 1.83 (1.42, 2.36) <b>P per trend &lt;0.001</b>  <b>Model 3; RR (95% CI)</b> <u>C4:</u> 1.39 (1.07, 1.76) <b>P per trend &lt;0.01</b>  <b>Model 4; RR (95% CI)</b> <u>C4:</u> 1.32 (1.01, 1.73) <b>P per trend &lt;0.04</b>  <b>A positive (non-significant) association was observed for ASB</b> <b>Model 3; RR (95% CI)</b> <u>C4 vs C1:</u> 1.21 (0.97, 1.50) <b>P per trend = 0.12</b> <i>RR remained unchanged after additional adjustment for energy intake</i>
3	<b>MDCS</b>  Sweden  Ericson et al. (2018)*  18.4 y (mean)  Public funding	<b>Same population and exclusion criteria as for added sugars</b>	<b>Same ascertainment of the outcome as for added sugars</b>	<b>g/d (range)</b> <u>Non-consumers (ref):</u> 0 <u>Tc1:</u> 0.3-47.1 <u>Tc2:</u> 47.3-142.8 <u>Tc3:</u> 142.9-3,000  <b>n/person-years</b> <u>Non-consumers (ref):</u> 12,066/221,229 <u>Tc1:</u> 5,103/95,790 <u>Tc2:</u> 4,596/85,689 <u>Tc3:</u> 4,857/86,478  <b>Exposure assessment:</b> 7-d food record and SFFQ	<u>Non-consumers (ref):</u> 1746 <u>Tc1:</u> 749 <u>Tc2:</u> 723 <u>Tc3:</u> 828	<b>Model 1:</b> sex, age, diet-method version, season, and total energy intake  <b>Model 2:</b> model 1 + physical activity, alcohol intake, smoking, and education  <b>Model 3:</b> model 2 + BMI  <b>Model 4:</b> model 3 + coffee, meat, whole grains	<b>Model 1; HR (95%CI)</b> <u>Non-consumers (ref):</u> 1 <u>Tc1:</u> 1.02 (0.94, 1.11) <u>Tc2:</u> 1.10 (1.01, 1.20) <u>Tc3:</u> 1.21 (1.12, 1.32) <b>P per trend &lt; 0.001</b>  <b>Model 2; HR (95%CI)</b> <u>Non-consumers (ref):</u> 1 <u>Tc1:</u> 1.02 (0.94, 1.12) <u>Tc2:</u> 1.09 (1.00, 1.19) <u>Tc3:</u> 1.14 (1.05, 1.25) <b>P per trend = 0.001</b>  <b>Model 3; HR (95%CI)</b> <u>Non-consumers (ref):</u> 1 <u>Tc1:</u> 1.03 (0.94, 1.12) <u>Tc2:</u> 1.06 (0.97, 1.15) <u>Tc3:</u> 1.06 (0.97, 1.16)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
							<b>P per trend = 0.123</b>  <b>Model 4; HR (95%CI)</b> Non-consumers (ref): 1 Tc1: 1.02 (0.94, 1.12) Tc2: 1.05 (0.96, 1.15) Tc3: 1.05 (0.96, 1.14) <b>P per trend = 0.228</b>
<b>Exposure: SSSD + SSFD + SSFJ</b>							
<b>1</b>	<b>Toyama</b>  Japan  Sakurai et al. (2014)  7 y  Public funding	<b>N = 2,275</b>  <b>Population sampled:</b> employees of a factory  <b>Excluded:</b> cases of diabetes or high levels of fasting plasma glucose or glycated haemoglobin at baseline; total daily energy intake < 500 kcal or > 5,000 kcal; SSSD consumption data unavailable, loses to follow-up.  <b>n = 2,037</b>  <b>Sex:</b> males <b>Ethnicity:</b> Asian <b>Age:</b> 35-55 y	Fasting plasma glucose and HbA1c measured during the annual medical examinations. According to the definition of the ADA <sup>30</sup> and the JDS <sup>31</sup> , diagnosis confirmed by at least one of the following observations: fasting plasma glucose concentration ≥126 mg/dl; HbA1c value ≥6.5%; treatment with insulin or oral hypoglycaemic agent.	<b>Servings/d Median (IQR)</b>  C1 (ref): 0 C2: 0.12 (0.12-0.21) C3: 0.48 (0.30-0.84) C4: 2.1 (1.4-2.7)  Serving size = 8 oz (237 mL)  <b>n/person-years</b> C1 (ref): 660/3,554 C2: 271/ 1,494 C3: 865/ 4,825 C4: 241/ 1,381  <b>Exposure assessment:</b> SFFQ	C1 (ref): 55 C2: 19 C3: 72 C4: 24	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + BMI  <b>Model 3:</b> model 2 + family history of diabetes, smoking, alcohol, habitual exercise, hypertension, dyslipidaemia, diet treatment for chronic disease, total energy intake and total fibre intake  <b>Model 4:</b> model 3 + consumption of ASSD, FJ, vegetable juice and coffee  <b>Adjustments as specified in Models 2 and 3 did not materially change the RRs as estimated in Model 4 (not shown)</b>	<b>Model 1; HR (95% CI)</b> C1 (ref): 1 C2: 0.86 (0.51, 1.45) C3: 1.03 (0.72, 1.46) C4: 1.24 (0.77, 2.01) <b>P per trend = 0.296</b>  <b>Model 4; HR (95% CI)</b> C1 (ref): 1 C2: 0.97 (0.57, 1.64) C3: 1.11 (0.74, 1.66) C4: 1.34 (0.72, 2.36) <b>P per trend = 0.424</b>  <b>A stronger positive (significant) association was observed for ASB</b> <b>Model 4; HR (95% CI)</b> C3 vs C1: 1.71 (1.11, 2.63) <b>P per trend = 0.015</b> Only 3 categories of intake for ASB as very few consumed ≥1 serving/d.

<sup>30</sup> Sakurai M, Nakamura K, Miura K, Takamura T, Yoshita K, Nagasawa SY et al (2012) Self-reported speed of eating and 7-year risk of type 2 diabetes mellitus in middle-aged Japanese men. Metabolism 61:1566-1571

<sup>31</sup> The committee of the Japan Diabetes Society on the diagnostic criteria of diabetes mellitus (2012) Report of the Committee on the classification and the diagnostic criteria of diabetes mellitus. J Diabetes Invest 3: 39-40

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
							<i>C3 for ASB is <math>\geq 1</math> serving/week</i>
<b>2</b>	<b>JPHC</b>  Japan  Eshak et al. (2013)  Up to 10 y  Public funding	<b>N</b> = 43,149  <b>Population sampled:</b> general population from 5 prefectures  <b>Excluded:</b> self-reported diabetes, CVD, cancer, kidney disease or chronic liver disease at baseline; missing baseline data for any of the exposure parameters: SSSD, 100% FJ and vegetable juice intake; implausible total energy intake (<500 or >3500 kcal/d)  <b>Follow-up rate males:</b> 70.5% <b>Follow-up rate females:</b> 78.2%  <b>n</b> = 27,585 Males: 12,137 Females: 15,448 <b>Ethnicity:</b> Asian <b>Age:</b> 40-59 y	Self-reported, positive response to the question "has a doctor ever told you that you had diabetes? In any of the follow-up health questionnaire (at 5 and/or 10 y).  All incident cases were classified as T2DM because the age of onset in the cohort was > 40 years.  <b>Positive predictive value for incident T2DM</b> = 94% as compared with medical records; 98% as compared to measured glucose and HbA <sub>1c</sub>  <b>Negative predictive value when diabetes is not reported</b> = 95% as compared to measured glucose and HbA <sub>1c</sub>  <b>Sensitivity = 46%</b> in a validation study <sup>32</sup> using the WHO (1985) criteria and the ADA (1997) criteria	<b>servings/week (range)</b>  <u>C1 (ref):</u> 0 <u>C2:</u> $\leq 2$ <u>C3:</u> 3-4 <u>C4:</u> 5-7  Serving size = 250 g  <b>n</b> <b>Men</b> <u>C1 (ref):</u> 6,155 <u>C2:</u> 3,326 <u>C3:</u> 1,597 <u>C4:</u> 1,059  <b>Women</b> <u>C1 (ref):</u> 10,121 <u>C2:</u> 3,408 <u>C3:</u> 1,198 <u>C4:</u> 721  <b>Exposure assessment:</b> SFFQ	<b>Men</b> <u>C1 (ref):</u> 261 <u>C2:</u> 121 <u>C3:</u> 58 <u>C4:</u> 44  <b>Women</b> <u>C1 (ref):</u> 200 <u>C2:</u> 83 <u>C3:</u> 30 <u>C4:</u> 27	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + BMI, family history of diabetes mellitus, education, occupation, smoking status, alcohol intake, history of hypertension, leisure-time physical activity, consumption of coffee, consumption of green tea, energy-adjusted intakes of dietary magnesium, calcium, vitamin D, rice and total dietary fibre and total energy intake	<b>Model 1; OR (95%CI)</b> <b>Men</b> <u>C1 (ref):</u> 1 <u>C2:</u> 0.86 (0.69, 1.07) <u>C3:</u> 0.86 (0.64, 1.15) <u>C4:</u> 1.00 (0.72, 1.39) <b>P per trend = 0.85</b>  <b>Women</b> <u>C1 (ref):</u> 1 <u>C2:</u> 1.27 (0.98, 1.65) <u>C3:</u> 1.31 (0.89, 1.93) <u>C4:</u> 1.97 (1.31, 2.97) <b>P per trend = 0.0005</b>  <b>Model 2; OR (95%CI)</b> <b>Men</b> <u>C1 (ref):</u> 1 <u>C2:</u> 0.86 (0.68, 1.08) <u>C3:</u> 0.83 (0.61, 1.12) <u>C4:</u> 0.98 (0.68, 1.42) <b>P per trend = 0.80</b>  <b>Women</b> <u>C1 (ref):</u> 1 <u>C2:</u> 1.15 (0.88, 1.51) <u>C3:</u> 1.17 (0.78, 1.76) <u>C4:</u> 1.79 (1.11, 2.89) <b>P per trend = 0.01</b>
<b>Exposure: SSSD + SSFD + TFJ</b>							
<b>1</b>	<b>ARIC</b>  USA	<b>N</b> = 15,792  <b>Population sampled:</b> general population from 4 US communities	T2DM at baseline was defined as the presence of any of the following criteria: fasting glucose of $\geq 126$ mg/dl, non-fasting glucose of $\geq 200$ mg/dl, current	<b>servings/d (range)</b>  <b>Men</b> <u>C1 (ref):</u> <1	<b>Men</b> <u>C1 (ref):</u> 331 <u>C2:</u> 67 <u>C3:</u> 182 <u>C4:</u> 138	<b>Model 1:</b> age, race, education, and family history of diabetes  <b>Model 2:</b> model 1 + BMI, waist:hip ratio, total energy intake, dietary	<b>Model 1; HR (95% CI)</b> <b>Men</b> <u>C1 (ref):</u> 1 <u>C2:</u> 1.03 (0.79, 1.34) <u>C3:</u> 0.95 (0.79, 1.15)

<sup>32</sup> Waki K, Noda M, Sasaki S, Matsumura Y, Takahashi Y, Isogawa A, et al. Alcohol consumption and other risk factors for self-reported diabetes among middle-aged Japanese: a population-based prospective study in the JPHC study cohort I. Diabetic Med 2005;22:323C31.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Paynter et al. (2006)  Up to 9 y  Public funding	<b>Excluded:</b> ethnicity other than black or white, diabetes or unknown diabetes status at baseline, missing exposure or diabetes information, missing information on any of the potential confounders, individuals not returning after baseline visit.  <b>n</b> = 12,204  Males = 5,414 Females = 6,790  <b>Ethnicity:</b> 78.1% White, 21.9% African American  <b>Age:</b> 45–64 y	use of hypoglycemic medication, positive response to the question “has a doctor ever told you that you had diabetes?”  Glucose values checked at clinic visits every 3 years.  Diagnosis of incident T2DM made using glucose values in ascertainment visits during follow-up using the criteria specified above.  Diagnosis based on self-reported only + medication use used for sensitivity analysis and comparability with other studies.	<u>C2</u> : 1 <u>C3</u> : 1.1-1.9 <u>C4</u> : ≥2.0  <b>Women</b> <u>C1</u> (ref): <1 <u>C2</u> : 1 <u>C3</u> : 1.1-1.9 <u>C4</u> : ≥2.0  Serving size = 8oz (240 mL)  <b>n/ person-years</b> <b>Men</b> <u>C1</u> (ref): 2,557/ 19,205 <u>C2</u> : 504/ 3,706 <u>C3</u> : 1,415/ 10,665 <u>C4</u> : 938/ 6,892  <b>Women</b> <u>C1</u> (ref): 3,510/ 27,438 <u>C2</u> : 896/ 6,815 <u>C3</u> : 1,490/ 11,255 <u>C4</u> : 894/ 6,533  <b>Exposure assessment:</b> SFFQ	<b>Women</b> <u>C1</u> (ref): 320 <u>C2</u> : 103 <u>C3</u> : 182 <u>C4</u> : 114	fibre, smoking, alcohol consumption, leisure activity, and hypertension.  <b>Adjustments as specified in Model 2 did not materially change the RRs as estimated in Model 1 (data not shown in the publication)</b>	<u>C4</u> : 1.03 (0.82, 1.28) <b>P per tend = 0.94</b>  <b>Women</b> <u>C1</u> (ref): 1 <u>C2</u> : 1.13 (0.91, 1.42) <u>C3</u> : 1.10 (0.91, 1.33) <u>C4</u> : 1.01 (0.79, 1.29) <b>P per tend = 0.58</b>
<b>2</b>	<b>TLGS</b>  Iran  Mirmiran et al. (2015)  3.6 y (mean)	<b>N</b> = 15,005  <b>Population sampled:</b> general population from one district of Tehran  <b>Excluded:</b> incomplete dietary intakes or missing measures of	Blood samples were drawn after an overnight fast and analysed at the TLGS laboratory.  Incident <b>high fasting blood glucose</b> was defined as ≥100 mg/dl or drug treatment during follow-up (survey 4).	<b>mL/d (median)</b> <u>Q1</u> (ref): 9.3 <u>Q2</u> : 32.0 <u>Q3</u> : 58.6 <u>Q4</u> : 142.2  <b>N of subjects per quartile for this outcome</b> NR	<b>NR</b>	<b>Model 1:</b> age, sex, total energy intake, physical activity and family history of diabetes  <b>Model 2:</b> model 1 + dietary fibre, tea and coffee, red a processed meat, fruit and vegetables  <b>Model 3:</b> model 2 + BMI	<b>Model 1; OR (95%CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 1.22 (0.56, 3.22) <u>Q3</u> : 1.90 (0.76, 4.72) <u>Q4</u> : 2.07 (0.79, 5.39) <b>P per trend: 0.079</b>  <b>Model 2; OR (95%CI)</b> <u>Q1</u> (ref): 1



RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Public funding	MetS components, reported energy intakes to energy requirements ratio beyond $\pm 3SD$ ; high FPG at baseline (survey 3).  <b>Follow-up rate:</b> 86%  <b>n</b> = 476  <b>Sex:</b> 68 % females <b>Ethnicity:</b> Caucasian <b>Age:</b> 6-18 y		<b>Exposure assessment:</b> SFFQ			<u>Q2:</u> 1.17 (0.44, 3.08) <u>Q3:</u> 1.83 (0.73, 4.58) <u>Q4:</u> 1.90 (0.71, 5.09) <b>P per trend: 0.109</b>  <b>Model 3; OR (95%CI)</b> <u>Q1</u> (ref): 1 <u>Q2:</u> 1.21 (0.48, 3.21) <u>Q3:</u> 1.87 (0.75, 4.68) <u>Q4:</u> 1.95 (0.73, 5.22) <b>P per trend: 0.108</b>
3	<b>WHI</b>  USA  Huang et al. (2017)  8.4 y (average)  Public funding	<b>N</b> = 122,970  <b>Population sampled:</b> Postmenopausal women recruited from 40 clinical centres  <b>Excluded:</b> prevalent DM cases at baseline and before or at AV3; ASB consumption not measured at the AV3; follow-up length not available; implausible dietary data (energy intake <600 or >5000 kcal/d); underweight; missing BMI; missing important covariates.  <b>n</b> = 64,850  <b>Sex:</b> females <b>Ethnicity:</b> ~ 84% Caucasian, 7.6% Black,	<b>Same ascertainment of outcome as for total sugars</b>	<b>Range (servings/time)</b>  <u>C1</u> (ref): <1/wk <u>C2:</u> 1-<7/wk <u>C3:</u> 1-<2 /d <u>C4:</u> $\geq 2/d$  Serving size = 12oz (355mL)  <b>Exposure assessment:</b> SFFQ	<b>NR</b>	<b>Model:</b> age, race, marital status, family income, education, family history of diabetes, BMI, change in BMI, waist-to-hip ratio, systolic blood pressure, insurance status, antihypertensive use, antihyperlipidemic use, hormone replacement therapy use, calibrated energy, SSSD consumption, glycemic load, glycemic index, Alternate Healthy Eating Index, cardiovascular history, hysterectomy history, smoking status, physical activity, sitting time and alcohol consumption.	<b>HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2:</u> 1.05 (0.98, 1.12) <u>C3:</u> 1.09 (0.97, 1.23) <u>C4:</u> 1.43 (1.17, 1.75) <b>P per trend = 0.0004</b>  <b>A positive (significant) association was observed for ASB</b> <b>HR (95% CI)</b> <u>C4 vs C1:</u> 1.21 (1.08, 1.36) <b>P per trend &lt;0.0001</b> <i>C1 for ASB is never or &lt;3 servings/month.</i> <i>Relationship only significant in the obese in subgroup analysis</i>



RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		Hispanic/Latino 4% and 3% Asian/Pacific <b>Age:</b> 50-79 y					
<b>Exposure: TFJ</b>							
<b>2</b>	<b>EPIC-InterAct<sup>33</sup></b>  FR, UK, NL, DE, DK, SE, IT, ES  InterAct consortium (2013)*  16 y  <b>Prospective case-cohort</b>  Public funding	<b>N</b> = 29,238  <b>Population sampled:</b> Mainly general population recruited from 8 EU countries  <b>Excluded:</b> diabetes at baseline, within the lowest and highest 1% of the cohort distribution of the ratio of reported total energy intake:energy requirement, with missing information on diet, physical activity, level of education, smoking status or BMI.  <b>n</b> = 27,058  <b>Random sub-cohort</b> n = 15,374 <b>Incident T2DM cases</b> n = 11,684  <b>Sex:</b> 62.5% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 35-70 y	<b>Same ascertainment of outcome as for SSSD+SSFD</b>	<b>Median, g/d (Servings/time, range)</b>  <u>C1</u> (ref): 0(<1/mo) <u>C2</u> : 17.1 (1-4/mo) <u>C3</u> : 100 (>1-6/wk) <u>C4</u> : 338.3 (≥1/d)  <b>Serving size</b> = 250 g  <b>n/category of intake:</b>  <u>C1</u> : 12,569 <u>C2</u> : 3,957 <u>C3</u> : 8,186 <u>C4</u> : 1,616  <b>Exposure assessment:</b> SFFQ	<u>C1</u> (ref): 5,837 <u>C2</u> : 1,702 <u>C3</u> : 3,425 <u>C4</u> : 720	<b>Model 1:</b> crude  <b>Model 2:</b> sex, educational level, physical activity, smoking status and alcohol consumption; total soft drinks (SSBs and ASBs)  <b>Model 3:</b> Model 2 + energy intake  <b>Model 4:</b> Model 3 + BMI	<b>Model 1; HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 0.88 (0.80, 0.98) <u>C3</u> : 0.89 (0.83, 0.94) <u>C4</u> : 0.97 (0.85, 1.11) <b>P for trend</b> = <b>0.64</b>  <b>Model 2; HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 0.91 (0.80, 1.02) <u>C3</u> : 0.96 (0.88, 1.04) <u>C4</u> : 1.00 (0.87, 1.15) <b>P for trend</b> = <b>0.63</b>  <b>Model 3; HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 0.91 (0.81, 1.02) <u>C3</u> : 0.96 (0.88, 1.04) <u>C4</u> : 0.99 (0.86, 1.14) <b>P for trend</b> = <b>0.84</b>  <b>Model 4; HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 0.97 (0.86, 1.10) <u>C3</u> : 1.04 (0.96, 1.13) <u>C4</u> : 1.06 (0.90, 1.25) <b>P for trend</b> = <b>0.21</b>  <b>HR (95% CI) per each 336 g increment</b> <u>M1</u> : 1.00 (0.92, 1.10) <u>M2</u> : 1.04 (0.96, 1.14) <u>M3</u> : 1.02 (0.94, 1.12) <u>M4</u> : 1.05 (0.94, 1.18)

<sup>33</sup> Data from individual countries was used for the dose-response meta-regression analysis as provided by the authors

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
2	<b>SUN</b>  Spain  Fresan et al. (2017)*  10.2 y (median)  Public funding	<b>N</b> = 21,678  <b>Population sampled:</b> University graduates, mainly health professionals  <b>Excluded:</b> participants susceptible of developing T2DM had prevalent T2DM, implausible energy intake, missing follow-up questionnaires, those reporting less than two servings/week of liquids, and those not answering more than 9/18 beverage items in the FFQ and drank less than one serving/day of beverages  <b>Follow-up rate:</b> 91.6%  <b>n</b> = 17,518  <b>Sex:</b> 60.43% females <b>Ethnicity:</b> Caucasian <b>Age:</b> ≥18 y	Incident T2DM was defined as those participants who were free of diabetes at baseline and reported a diagnosis by a doctor at follow-up.  Participants answered to an additional confirmation questionnaire and their medical records were requested. An endocrinologist blind to the exposure confirmed incident cases, according to American Diabetes Association <sup>34</sup>	<b>Servings/day</b> <b>C1 (ref):</b> 0 <b>C2:</b> <1 <b>C3:</b> 1-3 <b>C4:</b> >3  <b>Serving size = 200 ml</b>  <b>Mean ml/d</b> <b>C1 (ref):</b> 0 <b>C2:</b> 56 <b>C3:</b> 238 <b>C4:</b> 796  <b>n per category of intake</b> <b>C1 (ref):</b> 3,122 <b>C2:</b> 10,803 <b>C3:</b> 3,395 <b>C4:</b> 198 <b>Person-years per category of intake</b> <b>C1 (ref):</b> 29,712 <b>C2:</b> 103,977 <b>C3:</b> 32,262 <b>C4:</b> 1,804  <b>Exposure assessment:</b> SFFQ	<b>Cases per category of intake</b> <b>C1 (ref):</b> 40 <b>C2:</b> 72 <b>C3:</b> 28 <b>C4:</b> 2  <b>Total cases</b> = 142	<b>Model:</b> Sex, age, baseline BMI, familiar diabetes history, smoking habit, adherence to the Mediterranean dietary pattern, physical activity, time spent in sedentary activities, prevalent hypertension, servings/day of sugar-sweetened sodas, snacking between meals and total energy intake from other sources than TFJ.	<b>HR (95% CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 0.90 (0.61, 1.34) <b>C3:</b> 0.99 (0.60, 1.63) <b>C4:</b> 0.82 (0.20, 3.42) <b>P for trend = 0.862</b>
<b>Exposure: 100% FJ</b>							
1	<b>BWHS</b>  USA	<b>N</b> = 59,000  <b>Population sampled:</b> same as for SSSD	<b>Same as for SSSD</b>	<b>Servings/time (range)</b> <b>C1:</b> <1/mo <b>C2:</b> 1-7/mo <b>C3:</b> 2-6/wk	<b>C1 (ref):</b> 441 <b>C2:</b> 767 <b>C3:</b> 891 <b>C4:</b> 445 <b>C5:</b> 147	<b>Model 1:</b> age (only IRR and not 95%CI given for this model)  <b>Model 2:</b> model 1 + family history of diabetes, physical activity,	<b>Model 1; IRR</b> <b>C1 (Ref):</b> 1 <b>C2:</b> 0.92 <b>C3:</b> 0.96 <b>C4:</b> 0.93

<sup>34</sup> American Diabetes Association. Classification and diagnosis diabetes. Diabetes Care 2015 Jan;38(Suppl.1):S8e16.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Palmer et al. (2008)  10 y  Public funding	<b>Excluded:</b> same as for SSSD  <b>n</b> = 43,960  <b>Sex:</b> females <b>Ethnicity:</b> African American <b>Age:</b> 21-69 y		<u>C4:</u> 1/d <u>C5:</u> ≥2/d  Serving size = 6 oz (168 g)  <b>Person-years</b> <u>C1 (ref):</u> 50,871 <u>C2:</u> 102,984 <u>C3:</u> 111,975 <u>C4:</u> 53,789 <u>C5:</u> 16,620  <b>Exposure assessment:</b> SFFQ		cigarette smoking, years of education and each of the 2 other types of drinks (SSSD and SSSD/SSFJ)  <b>Model 3:</b> model 2 + intake of red meat, processed meats, cereal fibre and coffee and GI	<u>C5:</u> 1.02  <b>Model 2; IRR (95%CI)</b> <u>C1 (Ref):</u> 1 <u>C2:</u> 0.94 (0.84, 1.06) <u>C3:</u> 1.00 (0.89, 1.13) <u>C4:</u> 1.00 (0.88, 1.15) <u>C5:</u> 1.10 (0.91, 1.33)  <b>Model 3; IRR (95%CI)</b> <u>C1 (Ref):</u> 1 <u>C2:</u> 0.93 (0.83, 1.05) <u>C3:</u> 0.99 (0.88, 1.11) <u>C4:</u> 0.99 (0.87, 1.14) <u>C5:</u> 1.11 (0.92, 1.35) <b>P per trend = 0.28</b>
1	HPFS  USA  Muraki et al. (2013)*  Up to 22 y  Public funding	<b>N</b> = 51,529  <b>Population sampled:</b> male health professionals  <b>Excluded:</b> diagnosis of diabetes, CVD or cancer at baseline; missing data for individual fruits and fruit juice; unusual level of total energy intake (<800 or >4,200 kcal/d), diagnosis of T2DM unclear; completed baseline questionnaire only.  <b>n</b> = 36,173  <b>Sex:</b> males <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 40-75 y	<b>Same ascertainment of outcome as for SSSD + SSFD</b>	<b>servings/time (range)</b> <u>C1 (ref):</u> <1/wk <u>C2:</u> 1/wk <u>C3:</u> 2-4/wk <u>C4:</u> 5-6/wk <u>C5:</u> 3 1/d  Serving size= 6 oz (168 g)  <b>Person-years</b> <u>C1 (ref):</u> 93,948 <u>C2:</u> 49,856 <u>C3:</u> 119,407 <u>C4:</u> 112,021 <u>C5:</u> 279,172  <b>Exposure assessment:</b> SFFQ	<u>C1 (ref):</u> 401 <u>C2:</u> 225 <u>C3:</u> 488 <u>C4:</u> 460 <u>C5:</u> 1,113	<b>Model 1:</b> age, ethnicity, BMI, smoking status, multivitamin use, physical activity, family history of diabetes, total energy intake, modified alternate healthy eating index score, and total whole fruit consumption	<b>HR (95% CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 1.07 (0.91, 1.26) <u>C3:</u> 0.99 (0.86, 1.13) <u>C4:</u> 1.05 (0.92, 1.20) <u>C5:</u> 1.13 (1.01, 1.27)  <b>HR (95%CI) per each 3 servings/week change</b> 1.06 (1.01, 1.11)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
1	<b>WHI</b>  USA  Auerbach et al. (2017)  7.8 y (mean)  Public funding	<b>N</b> = 122,970  <b>Population sampled:</b> Postmenopausal women recruited from 40 clinical centres  <b>Excluded:</b> energy intake outliers on baseline FFQ ( $\leq 600$ kcal/d or $\geq 5000$ kcal/d); baseline self-reported past or current diabetes; missing answers to the two 100% FJ questions on the FFQ  <b>n</b> = 114,219  <b>Sex:</b> females <b>Ethnicity:</b> ~ 84% Caucasian, 7.6% Black, Hispanic/Latino 4% and 3% Asian/Pacific <b>Age:</b> 50-79 y	Participants were considered to have T2DM if they initiated medication to treat it.  80% of the participants self-reporting treatment for diabetes at baseline had diabetes medication in their medical inventory.  100% of participants that did not report diabetes treatment, had no diabetes medication in their baseline inventory.	<b>oz/d†</b> <b>Median (range)</b> <b>Q1 (ref):</b> 0 <b>Q2:</b> 1.0 (0.06-1.7) <b>Q3:</b> 2.7 (1.8-3.8) <b>Q4:</b> 5.1 (3.9-6.5) <b>Q5:</b> 8.0 (6.6-36.8)  1 oz @ 29.6 mL  <b>n/ person-years</b> <b>Q1 (ref):</b> 14,008/ 102,874 <b>Q2:</b> 25,053/ 183,543 <b>Q3:</b> 25,053/ 183,980 <b>Q4:</b> 25,053/ 183,210 <b>Q5:</b> 25,052/ 184,126  <b>Exposure assessment:</b> SFFQ	<b>Q1 (ref):</b> 1,435 <b>Q2:</b> 2,529 <b>Q3:</b> 2,522 <b>Q4:</b> 2,541 <b>Q5:</b> 2,461	<b>Model 1:</b> age, education level, race/ethnicity, smoking status, physical activity, BMI, hormone replacement therapy status, study arm and total energy intake.	<b>HR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 0.98 (0.92, 1.04) <b>Q3:</b> 0.99 (0.93, 1.05) <b>Q4:</b> 1.00 (0.93, 1.07) <b>Q5:</b> 0.97 (0.91, 1.03) <b>P per trend 0.17</b>
2	<b>CARDIA</b>  USA  Duffey et al. (2010)  20 y  Mixed funding	<b>Same population and exclusion criteria as for SSSD+SSFD</b>	<b>Same ascertainment of the outcome as for SSSD+SSFD</b>	<b>Kcal/day (mean±SEM)</b>  <b>Year 0;</b> n=5,034 115±2  <b>Year 7;</b> n= 3,877 114±9  Average of intake at 0 and 7 years used for the analysis = NR	267	<b>Model:</b> race, gender, centre, age, weight, smoking status, energy from food, total physical activity, energy from other beverages (low-fat milk, whole-fat milk and fruit juice), and energy from alcohol.	<b>Per 100 kcal increase* HR (95% CI)</b> 1.01 (0.91, 1.13)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
				Exposure assessment: SFF Q			
2	NHS  USA  Muraki et al. (2013) *  Up to 24 y  Public funding	N = 121,770  <b>Population sampled:</b> female nurses  <b>Excluded:</b> diagnosis of diabetes (including GDM), CVD or cancer at baseline; missing data for individual fruits and fruit juice; unusual level of total energy intake (<500 or >3,500 kcal/d); completed baseline questionnaire only.  n = 66,105  <b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~93%+) <b>Age:</b> 30-55 y	Same ascertainment of outcome as for the NHS II	<b>servings/time (range)</b> C1 (ref): <1/wk C2: 1/wk C3: 2-4/wk C4: 5-6/wk C5: 3 1/d  Serving size= 6 oz (168 g)  <b>Person-years</b> C1 (ref): 210,618 C2: 114,927 C3: 263,597 C4: 240,853 C5: 564,132  <b>Exposure assessment:</b> SFFQ	C1 (ref): 921 C2: 547 C3: 1,260 C4: 1,090 C5: 2,540	<b>Model 1:</b> age, ethnicity, BMI, smoking status, multivitamin use, physical activity, family history of diabetes, menopausal status and post-menopausal hormone use, total energy intake, modified alternate healthy eating index score, and total whole fruit consumption	<b>HR (95% CI)</b>  C1 (ref): 1 C2: 1.09 (0.98, 1.21) C3: 1.13 (1.03, 1.23) C4: 1.13 (1.03, 1.24) C5: 1.21 (1.12, 1.31)  <b>HR (95%CI) per each 3 servings/week change</b> 1.07 (1.04, 1.11)
2	NHS II  USA  Muraki et al. (2013) *  Up to 18 y  Public funding	N = 116,671  <b>Population sampled:</b> female nurses  <b>Excluded:</b> same as for the NHS above  n = 85,104  <b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~90%+)	Same ascertainment of outcome as for SSSD + SSFD	<b>servings/time (range)</b> C1 (ref): <1/wk C2: 1/wk C3: 2-4/wk C4: 5-6/wk C5: 3 1/d  Serving size= 6 oz (168 g)  <b>Person-years</b> C1 (ref): 248,276	C1 (ref): 672 C2: 357 C3: 777 C4: 494 C5: 853	<b>Model 1:</b> age, ethnicity, BMI, smoking status, multivitamin use, physical activity, family history of diabetes, menopausal status and post-menopausal hormone use, oral contraceptive use, total energy intake, modified alternate healthy eating index score, and total whole fruit consumption	<b>HR (95% CI)</b>  C1 (ref): 1 C2: 0.92 (0.81, 1.05) C3: 0.97 (0.87, 1.07) C4: 0.97 (0.86, 1.09) C5: 1.14 (1.02, 1.27)  <b>HR (95%CI) per each 3 servings/week change</b> 1.07 (1.02, 1.11)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		Age: 24-44 y		C2: 150,182 C3: 338,127 C4: 254,371 C5: 425,155  Exposure assessment: SFFQ			
3	JPHC  Japan  Eshak et al. (2013)  Up to 10 y  Public funding	N = 43,149  <b>Population sampled:</b> Same as for SSSD + SSFD + FJ  <b>Excluded:</b> Same as for SSSD + SSFD + FJ (any type)  <b>Follow-up rate males:</b> Males: 70.5% Females: 78.2%  n = 27,585 Males: 12,137 Females: 15,448  <b>Ethnicity:</b> Asian <b>Age:</b> 40-59 y	Same ascertainment of outcome as for SSSD + FD + FJ (any type)	<b>servings/week (range)</b> C1 (ref): 0 C2: ≤2 C3: 3-4 C4: 5-7  Serving size = 250 g  n <b>Men</b> C1 (ref): 7,115 C2: 3,744 C3: 914 C4: 364  <b>Women</b> C1 (ref): 9,075 C2: 4,616 C3: 1,198 C4: 559  Exposure assessment: SFFQ	<b>Men</b> C1 (ref): 302 C2: 129 C3: 36 C4: 17  <b>Women</b> C1 (ref): 198 C2: 99 C3: 25 C4: 18	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + BMI, family history of diabetes mellitus, education, occupation, smoking status, alcohol intake, history of hypertension, leisure-time physical activity, consumption of coffee, consumption of green tea, energy-adjusted intakes of dietary magnesium, calcium, vitamin D, rice and total dietary fibre and total energy intake	<b>Model 1; OR (95%CI)</b> <b>Men</b> C1 (ref): 1 C2: 0.81 (0.65, 0.99) C3: 0.92 (0.65, 1.31) C4: 1.10 (0.67, 1.82) <b>P per trend = 0.85</b>  <b>Women</b> C1 (ref): 1 C2: 0.98 (0.77, 1.25) C3: 0.94 (0.61, 1.42) C4: 1.45 (0.89, 2.37) <b>P per trend = 0.24</b>  <b>Model 2; OR (95%CI)</b> <b>Men</b> C1 (ref): 1 C2: 0.81 (0.65, 1.01) C3: 0.93 (0.65, 1.35) C4: 1.17 (0.69, 2.00) <b>P per trend = 0.94</b>  <b>Women</b> C1 (ref): 1 C2: 0.94 (0.73, 1.21) C3: 0.90 (0.58, 1.40) C4: 1.37 (0.79, 2.37) <b>P per trend = 0.41</b>

ADA, American Diabetes Association; ASB, artificially sweetened beverages; ASSD, artificially sweetened soft drinks; BMI, body mass index; CI, confidence interval; CHD, coronary heart disease; CVD, cardiovascular disease; d, day; DHI, dietary history interview; DGAI, Dietary Guidelines Adherence Index; DM, diabetes mellitus; EP, energy partition; ES, energy substitution; FD, fruit drinks; FFQ, food frequency questionnaire; FJ, fruit juice; FPG, fasting plasma glucose; GDM, gestational diabetes mellitus; GI, glycaemic index; HbA, haemoglobin A; HR, hazard ratio; IRR, incidence risk ratio;

mo, month; JDS, Japanese Diabetes Society; n, participants analysed; N, participants included in the cohort; NPV, negative predictive value; NR, not reported; OGTT, oral glucose tolerance test; PPV, positive predictive value; RR, risk ratio; SD, standard deviation; SFFQ, semiquantitative food frequency questionnaire; SSFD, sugar-sweetened fruit drinks; SSFJ, sugar-sweetened fruit juices; SSSD, sugar-sweetened soft drinks; T1DM, type 1 diabetes mellitus; T2DM, type 2 diabetes mellitus; TFJ, total fruit juices; TS, total sugars; UK, United Kingdom; USA, United States of America; WC, waist circumference; WHO, World Health Organization; wk, week; y, year. \*Data provided by authors. † Exposure adjusted for total energy intake using the nutrient residuals model. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Continuous measures of blood lipids

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
<b>Exposure: Total sugars</b>							
<b>2</b>	<b>BMES</b>  Australia  Goletzke et al. (2013a)  5 y  Public funding	<b>N</b> = 3,654  <b>Population sampled:</b> General population  <b>Excluded:</b> Incomplete or implausible dietary data (daily energy intakes <2500 or >18 000 kJ), and missing blood samples.  <b>n</b> = 755  <b>Follow-up rate:</b> 91%  <b>Sex:</b> 62.7% females <b>Ethnicity:</b> Caucasian <b>Age (median (IQR)):</b> 67 y (62, 73)	<b>TG and HDL-c</b>  <b>Fasting blood samples</b> were drawn, centrifuged on site and sent to another laboratory for analysis of blood lipids at baseline and follow-up.	<b>E% (median (IQR))</b> 25.2 (21.2, 29.2)  <b>Method:</b> SFFQ	<b>Changes in total sugars</b> intake vs changes in TG and HDL-c over the 5-7 follow-up.  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> sex, time defined as 1 (baseline) and 2 (5-y follow-up), baseline sugar intake, baseline x time and concurrent change, and energy intake.  <b>Model 2:</b> Model 1 + age, BMI, diabetes, smoking (past and/or concurrent), alcohol consumption, the use of cholesterol-lowering medication and dietary fat (E%) and fibre intake (g/MJ) from fruits as terms at baseline, baseline x time and concurrent change.	Non-significant ( <b>positive</b> ) associations were observed between changes in total sugar intake and concurrent changes in TG and HDL-c over the follow-up.  <b>Per each 1E% increase <math>\beta</math> coefficients (SE)</b>  <b>log TG</b> Model 1: 0.0008 (0.0022), <b>P</b> = 0.7 Model 2: 0.0022 (0.0028), <b>P</b> = 0.4  <b>log HDL-c</b> Model 1: 0.0001 (0.0010), <b>P</b> = 0.9 Model 2: 0.0011 (0.0013), <b>P</b> = 0.4
<b>1</b>	<b>ALSPAC</b>  UK  Cowin and Emmett (2001)*  13 mo  Public funding	<b>N</b> = 1,341  <b>Population sampled:</b> General population living within a defined part of the country  <b>Excluded:</b> twins, non-white children  <b>n per outcome</b> <b>T-c</b> Females: 175 Males: 214 <b>HDL-c</b> Females: 133 Males: 164 <b>LDL-c</b> Females: 109	<b>T-c, HDL-c, LDL-c and T:HDL-c</b>  At follow-up a <b>non-fasting</b> blood sample was obtained by venepuncture. <b>TG and T-c</b> were measured using standard enzymatic colorimetric tests. <b>HDL-c</b> was measured in the same way as T-c after precipitation of LDL-c using a Bayer kit. <b>LDL-c</b> was calculated using the Friedewald equation. When TG levels were >2	<b>g/d (mean (SE))</b> † Females: 72.3 (0.9) Males: 78.2 (0.9)  <b>Method</b> : 3-d DR	<b>Total sugars intake</b> at baseline vs T-c, HDL-c, LDL-c and T-c:HDL-c ratio at end of follow-up  <b>Data collection:</b> exposure at baseline and outcome at end of follow-up	<b>Model 1:</b> crude  <b>Model 2:</b> Multivariate regression models initially included energy and the energy-adjusted intakes of saturated, monounsaturated and polyunsaturated fat, cholesterol, starch, sugar and vitamin C. Backwards stepwise regression was used to exclude the least significant variable until all remaining had a P<0.10  <b>Model 3:</b> model 2 + birthweight	<b>Model 1; correlation coefficients</b> <b>T-c</b> Females: 0.009, <b>P</b> = 0.906 Males: 0.152, <b>P</b> = 0.026 <b>HDL-c</b> Females: -0.024, <b>P</b> = 0.784 Males: 0.030, <b>P</b> = 0.702 <b>LDL-c</b> Females: 0.053, <b>P</b> = 0.583 Males: -0.152, <b>P</b> = 0.076 <b>T:HDL-c</b> Females: 0.041, <b>P</b> = 0.642 Males: -0.142, <b>P</b> = 0.073  <b>Model 3</b> <b>Females:</b> sugars retained in the model for the T-c:HDL-c ratio only (positive association)



RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
		Males: 137  <b>Ethnicity:</b> Caucasian <b>Age:</b> 1.5 y	mmol/l, LDL-c was not calculated.			<b>Results for model 2 were as for model 3</b>	<b>Males:</b> sugars not retained in the model for any blood lipid variable
<b>Exposure: added sugars</b>							
<b>1</b>	<b>NGHS</b>  USA  Lee et al. (2014)  Up to 10 y  Unclear funding	<b>N= 2,379</b>  <b>Population sampled:</b> Non-Hispanic Caucasian and African American girls with racially concordant parents from 3 sites  <b>Excluded:</b> Hispanic or other races; implausible caloric intake of <650 calories or >4000 calories; missing non-fasting HDL-c, nutritional data and other covariates; pregnancy.  <b>n= 2,223 (6,837 observations)</b>  n at visit 1= 1,709 n at visit 3= 1,619 n at visit 5= 1,486 n at visit 7= 1,205 n at visit 10= 818  <b>Sex:</b> Females <b>Ethnicity:</b> 47% Black and 53% Caucasian <b>Age</b> (mean $\pm$ SD): 10 $\pm$ 0.6 y	<b>HDL-c</b>  <b>HDL-c levels measured in non-fasting blood samples.</b> For girls who had both non-fasting and fasting HDL-c measurements, the correlation between the 2 values was >0.99	<b>E%</b> <b>C1</b> <10% <b>C2:</b> $\geq$ 10%  <b>n at visit 1:</b> <b>C1:</b> 210 <b>C2:</b> 1,499 <b>n at visit 5:</b> <b>C1:</b> 169 <b>C2:</b> 1,317 <b>n at visit 10</b> <b>C1:</b> 86 <b>C2:</b> 732  <b>Method:</b> 3-d DR	<b>Added sugars</b> intake at baseline vs change in HDL-c over the 10-y follow-up  <b>Data collection:</b> Baseline (visit 1), every second year (visits 3, 5, and 7) and end of follow-up (visit 10)	<b>Model:</b> age, race, smoking, physical activity, puberty stage, BMI category, total energy, nutrient residuals for: fiber, other carbohydrates, saturated fat, monounsaturated fat, polyunsaturated fat, total energy and age, BMI category and age.  <i>Added sugars consumption was treated as a time-varying covariate</i>	<b>Significant positive association</b> between added sugar consumption of <10% and changes in HDL-c over the 10-y follow-up. Consumption of $\geq$ 10% added sugars was non-significantly (negative) associated with changes in HDL-c.  <b>Between-group adjusted difference in HDL-c change/year (mg/dL)</b> <b>Mean (95% CI)</b> C1 vs C2: 0.26 (0.04, 0.48), <b>P = 0.02</b>  <b>Predicted 10-y change in HDL-c (mg/dL)</b> <b>Mean (95% CI)</b> <b>C1:</b> 2.2 (0.09, 4.32), <b>P = 0.04</b> <b>C2:</b> -0.4 (-1.32, 0.52), <b>P = 0.4</b>
<b>Exposure: sucrose</b>							

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
2	<b>NSHDS</b>  Sweden  Winkvist et al. (2017)  10 y  Mixed funding	<b>N</b> = 40,066  <b>Population sampled:</b> General population  Excluded: Between visits interval <9y or >11y; >10% of the FFQ missing or missing portion sizes; implausible energy intakes, missing body weight; weight < 35 kg, length <130 cm or BMI <15 kg/m <sup>2</sup> .  <b>n</b> = 15,995 Females = 8,354 Males = 7,641  <b>Ethnicity:</b> Caucasian <b>Age:</b> 30 – 60 y	<b>T-c and TG</b>  <b>Fasting</b> venous blood samples were used for analysis of serum <b>cholesterol</b> and <b>triglycerides</b> . These were measured in health centers using a Reflotron bench top analyzer (1990-2009) or using an enzymatic routine method at hospital laboratories (2009-2014). Serum cholesterol and triglyceride values measured with Reflotron were calibrated to values corresponding to the enzymatic method.	<b>%E (mean ± SD)</b> Females: 6.5 ± 2.6 Males: 6.6 ± 2.9  <b>g/d (mean ± SD)</b> Females: 24.4 ± 12.6 Males: 32.2 ± 18.3  <b>Method:</b> SFFQ	<b>Changes in sucrose</b> intake vs changes in T-c and TG over the 10-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> baseline outcome variable, year of study participation, age, education, smoking status and physical activity at the beginning of the period  <b>Joint model i.e. whole grain, PUFA, cholesterol, trans-fatty acids and sucrose entered in the same model</b>	Non-significant associations between changes in sucrose intake and changes in T-c ( <b>positive</b> ) and TG ( <b>negative</b> ) over the follow-up in both sexes.  <b>Per each 1E% increase in intake β ± SE (mmol/l)</b>  <b>T-c</b> <u>Females:</u> 0.02 ± 0.02 <b>P = 0.43</b> <u>Males:</u> 0.001 ± 0.02 <b>P = 0.96</b>  <b>TG</b> <u>Females:</u> -0.019 ± 0.01 <b>P = 0.13</b> <u>Males:</u> -0.008 ± 0.02 <b>P = 0.60</b>
2	<b>CARDIA</b>  USA  Archer et al. (1998)  7 y  Public funding	<b>N</b> = 5,115  <b>Population sampled:</b> general population of 4 centres selected to balance subgroups of race, sex, education and age  <b>Excluded:</b> incomplete data at baseline or year 7, prevalent diabetes, implausible energy intakes (≤ 3.3 or ≥ 33.3 MJ for men and ≤ 2.5 or ≥ 25 MJ for women), and missing data for covariates used in the analyses.  <b>n</b> = 3,335 Black men: 670	<b>HDL-c</b>  Blood for measurement of lipids was drawn from seated participants into evacuated tubes coated with EDTA. Total <b>HDL-c</b> was determined by the method of Warnick et al. 1982 & 1986.	<b>%E (mean ± SD)<sup>35</sup></b> Black men: 7.96 ± 5.23 White men: 7.13 ± 5.28 Black women: 9.39 ± 6.67 White women: 6.88 ± 6.03  <b>Method:</b> SFFQ	<b>Changes in sucrose</b> intake vs changes in HDL-c over the 7-y follow-up.  <b>Note:</b> TG measured but considered only for adjustment in sensitivity analyses, not as an outcome.  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> baseline age and changes in: BMI, alcohol intake, smoking, physical activity.	<b>Significant negative</b> association between changes in sucrose and changes in HDL-c in white men, white women and black women over the follow-up.  <b>Per each 10% E increase in dietary sucrose</b>  <b>β coefficients (SE) (mmol/L)</b> <u>Black men:</u> -0.03 (0.02) <u>White men:</u> -0.04 (0.01) <b>p</b> < 0.01 <u>Black women:</u> -0.03 (0.01) <b>p</b> < 0.05 <u>White women:</u> -0.04 (0.01) <b>p</b> < 0.01

<sup>35</sup> Sucrose intake for the purpose of these analyses included added sucrose at baseline and added plus naturally occurring sucrose at year 7. Naturally occurring sucrose amounts were very small and thus the two estimates were comparable.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
		White men: 926 Black women: 842 White women: 897 <b>Age:</b> 18 – 30 y					
<b>Exposure: fructose</b>							
<b>3</b>	<b>TLGS</b>  Iran  Bahadoran et al. (2017)  6.7 y (mean)  Public funding	<b>N</b> = 15,005  <b>Population sampled:</b> general population from one district of Tehran  <b>Excluded:</b> Unusual energy intake (<800 kcal/day or >4200 kcal/day, respectively), or were on specific diets for hypertension, diabetes or dyslipidemia; those with a history of CVD at baseline.  <b>n</b> = 2,369  <b>Follow-up rate:</b> 99.5% <b>Sex:</b> 56.5% females <b>Ethnicity:</b> Caucasian <b>Age (mean ± SD):</b> 38.1±13.3y	<b>TG and HDL-c</b>  Blood samples were collected after an <b>overnight fast</b>	<b>%E (mean ± SD)</b> 6.4 ± 3.7  <b>Method:</b> SFFQ	<b>Fructose</b> intake at baseline vs changes in TG and HDL-c over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> age	<b>Significant negative</b> association between baseline fructose intake and changes in HDL-c over the follow-up. Non-significant (positive) association between baseline fructose intake and changes in TG over the follow-up.  <b>Per each 1 %E increase β regression (95% CI) (mg/dL)</b>  <b>TG</b> 0.310 (-0.521, 1.145)  <b>HDL-c</b> -0.297 (-0.410, -0.184)
<b>Exposure: SSSD+SSFD</b>							
<b>1</b>	<b>Framingham Offspring+</b>  USA  Haslam et al. (2020)  Up to 23 y (Mean 12.5 y)	<b>N</b> = 5,124  <b>Population sampled:</b> Offspring of the original cohort of the Framingham Heart Study  <b>Excluded:</b> missing lipoprotein data, lipoprotein changes not within 4 SDs of mean change, implausible energy intake, incomplete FFQ  <b>n (No. of observations)</b> 3,124 (8,859)	<b>TG, LDL-c and HDL-c</b>  <b>Fasting blood samples</b> drawn for analysis of blood lipids	<b>Range (Servings/time)</b> <u>C1</u> (ref): <1/mo <u>C2</u> : 1-4/mo <u>C3</u> : 1-2/wk <u>C4</u> : 3-7/wk <u>C5</u> : >1/d  <b>No. of observations per category:</b> <u>C1</u> : 3,497 <u>C2</u> : 1,666 <u>C3</u> : 1,321	<b>Average SSSD+SSFD</b> intake of the two measurements within the examination intervals vs concurrent 4-y changes in TG, LDL-c and HDL-c  <b>Data collection:</b> baseline/first examination ('91-	<b>Model:</b> age, sex, total energy, education, current smoking status, physical activity index, BMI, alcohol, servings per day of vegetables, whole fruits, whole grains, nuts/seeds, and seafood, as well as percent energy from saturated fat and adjustment for LCSB, and 100% fruit juice	<b>Mean difference in 4-year changes Beta-coefficients (SE)</b>  <b>TG (mg/dL):</b> <u>C1</u> : reference <u>C2</u> : 1.8 (1.8) <u>C3</u> : 2.5 (2.0) <u>C4</u> : 4.9 (1.9) <u>C5</u> : 2.6 (2.9) <b>P per trend</b> = 0.03  <b>LDL-c (mg/dL):</b> <u>C1</u> : reference <u>C2</u> : -0.6 (0.7)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
	Public funding	<b>Sex:</b> 53.1% <b>Ethnicity:</b> Caucasian <b>Age (mean±SD):</b> 54.8 ± 9.8 y		<u>C4:</u> 1,705 <u>C5:</u> 674  <b>Servings/day Geometric mean (IQR)</b> <u>First examination:</u> 0.09 (0.49) <u>Second examination:</u> 0.09 (0.44) <u>Third examination:</u> 0.08 (0.42) <u>Fourth examination:</u> 0.05 (0.20)  <b>Serving size =</b> 355 ml  <b>Method:</b> SFFQ	'95), second examination ('95-'98), third examination ('98-'01), fourth examination ('05-'08), and end-of-follow-up/fifth examination ('11-'14)		<u>C3:</u> 1.2 (0.8) <u>C4:</u> -0.004 (0.8) <u>C5:</u> 0.9 (1.2) <b>P per trend = 0.44</b>  <b>HDL-c (mg/dL):</b> <u>C1(ref):</u> 1 <u>C2:</u> -0.4 (0.3) <u>C3:</u> -0.5 (0.3) <u>C4:</u> -0.7 (0.3) <u>C5:</u> -1.8 (0.4) <b>P per trend = 0.0002</b>
1	<b>Framingham-3Gen<sup>+</sup></b>  USA  Haslam et al. (2020)  Up to 9 y (Mean 6.1 y)  Public funding	<b>N = 4,095</b>  <b>Population sampled:</b> a third generation of participants of the original cohort of the Framingham Heart Study  <b>Excluded:</b> missing lipoprotein data, lipoprotein changes not within 4 SDs of mean change, implausible energy intake, incomplete FFQ  <b>n = 2,800</b>  <b>Sex:</b> 54.3% females <b>Ethnicity:</b> Caucasian <b>Age (mean±SD):</b> 40.3 ± 8.8 y	<b>TG, LDL-c and HDL-c</b>  <b>Fasting blood samples</b> drawn for analysis of blood lipids	<b>Range (servings/time)</b> <u>C1(ref):</u> <1/mo <u>C2:</u> 1-4/mo <u>C3:</u> 1-2/wk <u>C4:</u> 3-7/wk <u>C5:</u> >1/d  <b>No. of observations per category:</b> <u>C1:</u> 867 <u>C2:</u> 549 <u>C3:</u> 483 <u>C4:</u> 576 <u>C5:</u> 325  <b>Servings/day</b>	<b>Average SSSD+SSFD</b> intake of the two measurements within the examination intervals vs concurrent 4-y changes in TG, LDL-c and HDL-c  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> age, sex, total energy, education, current smoking status, physical activity index, BMI, alcohol, servings per day of vegetables, whole fruits, whole grains, nuts/seeds, and seafood, as well as percent energy from saturated fat and adjustment for LCSB, and 100% fruit juice	<b>Mean difference in 4-year changes Beta-coefficients (SE)</b>  <b>TG (mg/dL):</b> <u>C1:</u> reference <u>C2:</u> 4.5 (2.4) <u>C3:</u> 2.8 (2.6) <u>C4:</u> 5.6 (2.6) <u>C5:</u> 10.8 (3.5) <b>P per trend = 0.006</b>  <b>LDL-c (mg/dL):</b> <u>C1:</u> reference <u>C2:</u> 0.3 (0.8) <u>C3:</u> -0.3 (0.9) <u>C4:</u> 1.2 (0.9) <u>C5:</u> 2.4 (1.2) <b>P per trend = 0.08</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results	
				<b>Geometric mean (IQR)</b> 0.12 (0.56)  <b>Serving size =</b> 355 ml  <b>Method:</b> SFFQ			<b>HDL-c (mg/dL):</b> C1: reference C2: -0.1 (0.4) C3: -0.4 (0.4) C4: -1.0 (0.4) C5: -0.8 (0.5) <b>P per trend = 0.01</b>	
2	<b>Daily-D</b>  USA  Van Rompay et al. (2015)  1 y  Public funding	<b>N = 690</b>  <b>Population sampled:</b> General population from Boston area schools  <b>Excluded:</b> if total energy intake was <500 or >5000 kcal/d, having diabetes or missing baseline or 12-mo data on SSBs or blood lipids.  <b>n = 380</b>  <b>Sex:</b> 50.8% females <b>Ethnicity:</b> 45% Caucasian, 13% Black, 18% Hispanic, 9% Asian and 15% multi-racial/other <b>Age:</b> 8 – 15 y	<b>HDL-c and TG</b>  Blood was collected after an <b>overnight fast</b> .	<b>SSB intake categories at baseline (servings/wk (median))</b> C1: Non-consumer C2: >0 to <2 (1.2) C3: ≥2 to <7 (3.4) C4: ≥7 (10.6)  <b>n</b> C1: 13 C2: 135 C3: 186 C4: 46  <b>SSB categories by change of intake (servings/wk):</b> C1: ≥1 /wk decrease C2: no change C3: ≥1 /wk increase  <b>n</b> C1: 154 C2: 122 C3: 104	<b>SSSD+SSFD</b> intake at <b>baseline</b> and <b>changes in SSSD+SSFD</b> intake vs changes in TG and HDL-c over the 1 y follow-up.  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> crude  <b>Model 2:</b> baseline age, sex, race/ethnicity, baseline lipid concentration, baseline pubertal status, baseline BMI z score, baseline sedentary time, and mean intakes of total energy, fruits/vegetables, and discretionary solid fats.	Non-significant ( <b>positive</b> ) association between <b>baseline SSSD+SSFD</b> intake and changes in HDL-c and TG over the 1 y follow-up.  <b>By SSB intake category (mg/dL) mean ± SEM</b>  <b>HDL-c</b> <b>Model 1:</b> C1: 1.4 ± 2.2 C2: 3.2 ± 0.7 C3: 2.5 ± 0.6 C4: 3.3 ± 1.2 <b>P for trend = 0.76</b>  <b>Model 2:</b> C1: 0.8 ± 2.2 C2: 3.7 ± 0.7 C3: 2.7 ± 0.6 C4: 2.5 ± 1.3 <b>P for trend = 0.47</b>	<b>Significant (negative)</b> and non-significant (positive) association between <b>changes in SSSD+SSFD</b> intake and changes in HDL-c and TG, respectively, over the 1-y follow-up.  <b>By SSB intake change category (mg/dL) mean ± SEM</b>  <b>HDL-c</b> <b>Model 1:</b> C1: 4.1 ± 0.6 C2: 2.2 ± 0.7 C3: 1.5 ± 0.8 <b>P for trend = 0.02</b>  <b>Model 2:</b> C1: 4.6 ± 0.8 C2: 2.0 ± 0.8 C3: 1.5 ± 0.8 <b>P for trend = 0.02</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results	
				Serving size: 355 ml  Method: SFFQ			<b>TG</b> <b>Model 1:</b> C1: 11.8 ± 8.1 C2: 4.0 ± 2.5 C3: 3.5 ± 2.1 C4: 4.8 ± 4.3 <b>P for trend = 0.80</b>  <b>Model 2:</b> C1: 18.6 ± 8.1 C2: 4.5 ± 2.7 C3: 2.1 ± 2.2 C4: 3.8 ± 4.8 <b>P for trend = 0.26</b>	<b>TG</b> <b>Model 1:</b> C1: 3.2 ± 2.3 C2: 1.4 ± 2.6 C3: 8.6 ± 2.8 <b>P for trend = 0.16</b>  <b>Model 2:</b> C1: 2.2 ± 3.0 C2: 1.0 ± 2.9 C3: 7.9 ± 3.0 <b>P for trend = 0.19</b>
<b>Exposure: SSSD+SSFD+SSFJ</b>								
<b>1</b>	<b>WAPCS</b>  Australia  Ambrosini et al. (2013)  3 y  Unclear	<b>N</b> = 2,868  <b>Population sampled:</b> offspring from mothers from the Raine study  <b>Excluded:</b> Subjects who reported not fasting before venepuncture.  <b>n</b> = 1,124 females= 537 males= 587  <b>Ethnicity:</b> Caucasian <b>Age (mean ± SD):</b> 14.0 ± 0.2 y	<b>TG, HDL-c and LDL-c</b>  Fasting blood samples were used to assess <b>triglycerides and HDL-c</b> by, and to calculate standardized methods in a hospital laboratory. <b>LDL-c</b> concentrations were calculated (not specified how)	<b>g/d (range (mean ± SD))</b> T1 (ref): 0 – 130 (48 ± 39) T2: 130 – 329 (223 ± 59) T3: 331 – 2,876 (665 ± 351)  <b>n for those changing tertile of SSB intake = NR</b>  <b>Method:</b> SFFQ	<b>Changes in SSSD+SSFD+SSFJ intake vs percent of change in TG, HDL-c and LDL-c over the 3-y follow-up</b>  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> age, pubertal stage, physical fitness, dietary misreporting, maternal education, and family income  <b>Model 2:</b> Model 1 + BMI  <b>Model 3:</b> Model 2 + Healthy and Western dietary pattern scores	A significant <b>positive</b> association between change in SSSD+SSFD+SSFJ intake and change in TG (model 2) became <b>non-significant</b> (model 3) after adjusting for dietary patterns in both sexes. Significant (males) and non-significant (females) <b>negative</b> associations between change in SSSD+SSFD+SSFJ intake and change in HDL-c (model 2). Association became <b>non-significant</b> (model 3) in males after adjustment for dietary patterns. <b>Non-significant positive and negative</b> association in females and males, respectively, between change in SSSD+SSFD+SSFJ intake and change in LDL-c over the 3-y follow-up.	<b>Per each tertile of intake increase Δ% (95% CI) vs T1</b>  <b>Per each tertile of intake increase Δ% (95% CI) vs T1</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results	
							<b>Females - TG</b> <b>Model 1:</b> T2: 4.2 (-1.8, 10.3) T3: 10.8 (4.2, 17.3) <b>P for trend = 0.001</b>  <b>Model 2:</b> T2: 4.2 (-1.7, 10.2) T3: 7.0 (0.4, 13.5) <b>P for trend = 0.033</b>  <b>Model 3:</b> T2: 3.8 (-2.4, 9.9) T3: 6.2 (-1.2, 13.7) <b>P for trend = 0.09</b>	<b>Males - TG</b> <b>Model 1:</b> T2: 0 (-7.0, 7.0) T3: 10.4 (3.4, 17.5) <b>P for trend = 0.003</b>  <b>Model 2:</b> T2: -2.2 (-9.0, 4.6) T3: 8.4 (1.6, 15.3) <b>P for trend = 0.011</b>  <b>Model 3:</b> T2: -3.5 (-10.5, 3.5) T3: 6.7 (-0.8, 14.1) <b>P for trend = 0.06</b>
							<b>Females – HDL-c</b> <b>Model 1:</b> T2: -1.2 (-4.6, 2.2) T3: -5.1 (-8.9, -1.4) <b>P for trend = 0.01</b>  <b>Model 2:</b> T2: -1.2 (-4.5, 2.1) T3: -2.2 (-5.9, 1.5) <b>P for trend = 0.23</b>	<b>Males – HDL-c</b> <b>Model 1:</b> T2: 0.4 (-2.8, 3.6) T3: -3.8 (-7.1, -0.5) <b>P for trend = 0.017</b>  <b>Model 2:</b> T2: 1.2 (-1.9, 4.2) T3: -3.1 (-6.2, 0.1) <b>P for trend = 0.038</b>  <b>Model 3:</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results	
							<b>Model 3:</b> T2: -1.4 (-4.8, 1.9) T3: -3.1 (-7.2, 1.0) <b>P for trend = 0.14</b>  <b>Females – LDL-c</b> <b>Model 3:</b> T2: 0 (-4.2, 4.2) T3: 0.7 (-4.5, 5.9) <b>P for trend = 0.81</b>	T2: 1.9 (-1.2, 5.1) T3: -2.3 (-5.6, 1.1) <b>P for trend = 0.14</b>  <b>Males – LDL-c</b> <b>Model 3:</b> T2: -2.3 (-7.2, 2.7) T3: -3.9 (-9.3, 1.4) <b>P for trend = 0.15</b>

BMI, body mass index; CI, confidence interval; cm, centimetre; CVD, cardiovascular disease; d, day; FFQ, food frequency questionnaire; FJ, fruit juice; HDL-c, high-density lipoprotein cholesterol; HR, hazard ratio; IQR, interquartile range; kcal, kilocalories; kg, kilograms; kJ, kilojoules; LDL-c, low density lipoprotein cholesterol; MJ, megajoules; mo, months; n, participants analysed; N, participants included in the cohort; NR, not reported; OR, odds ratio; PUFA, polyunsaturated fatty acids; RR, relative risk; SD, standard deviation; SE, standard error; SFFQ, semiquantitative food frequency questionnaire; SSB, sugar-sweetened beverages; SSFD, sugar-sweetened fruit drinks; SSFJ, sugar-sweetened fruit juices; SSSD, sugar-sweetened soft drinks; T-c, total cholesterol; TFJ, total fruit juice; TG, triglycerides; UK, United Kingdom; USA, United States of America; y, years. \*Data provided by authors. † Exposure adjusted for total energy intake using the nutrient residuals model ‡ Study identified through an update of the literature search. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*



## Incidence of dyslipidaemia

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure n Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: SSSD</b>							
<b>3</b>	<b>KoGES</b>  South Korea  Kang and Kim (2017)  5.7 y (mean)  Public funding	<b>N = 10,030</b>  <b>Population sampled:</b> general population living in Ansan (urban) and Ansung (rural) areas  <b>Excluded:</b> refused to participate in follow-up visits, insufficient information, non-responders to dietary examination, prevalence of CVD or cancer, prevalent dyslipidaemia at baseline  <b>Follow-up rate:</b> 63.3 %  <b>n per outcome</b> <b>TG:</b> n = 5,144 Females: 2,929 Males: 2,215  <b>HDL-c:</b> n = 5,111 Females: 2,111 Males: 3,000  <b>Ethnicity:</b> Asian <b>Age:</b> 40-69 y	<b>Fasting concentrations</b> of TG and HDL-c in plasma were enzymatically measured.  <b>High TG:</b> $\geq 1.7$ mmol/l <b>Low HDL-c:</b> HDL-c $< 1.0$ mmol/l in men or $< 1.3$ mmol/l in women	<b>Servings/week (range)</b> C1(ref): Rarely or never C2: $< 1$ C3: $\geq 1$ to $< 4$ C4: $\geq 4$  <b>n per category, TG:</b> C1(ref): 2,251 C2: 1,912 C3: 842 C4: 139  <b>n per category, HDL-c:</b> C1(ref): 2,212 C2: 1,799 C3: 919 C4: 181  <b>Serving size:</b> 200 ml  <b>Method:</b> SFFQ	<b>High TG</b> C1(ref): 781 C2: 634 C3: 345 C4: 54  <b>Low HDL-c</b> C1(ref): 1,313 C2: 996 C3: 499 C4: 105	<b>Model:</b> age, income level, education level, alcohol consumption, smoking status, physical activity, BMI, energy intake, percentage of fat, fibre intake and the presence of diseases	<b>HR (95% CI)</b>  <b>High TG</b> C1(ref): 1 C2: 0.89 (0.80, 0.98) C3: 1.26 (1.10, 1.43) C4: 1.20 (0.91, 1.60) <b>P for trend = 0.87</b>  <b>Low HDL-c</b> <b>Model 1:</b> C1(ref): 1 C2: 0.90 (0.83, 0.98) C3: 1.05 (0.94, 1.17) C4: 1.17 (0.96, 1.44) <b>P for trend = 0.90</b>
<b>Exposure: SSSD+SSFD</b>							
<b>1</b>	<b>Framingham -Offspring<sup>‡</sup></b>  USA  Haslam et al. (2020)	<b>N = 5,124</b>  <b>Population sampled:</b> Offspring of the original cohort of the Framingham Heart Study	<b>Fasting blood samples</b> drawn for analysis of blood lipids  <b>High TG:</b> $\geq 175$ mg/dL <b>High LDL-c:</b> $\geq 160$ mg/dL or use of LDL	<b>Range (Servings/time)</b> C1(ref): $< 1$ /mo C2: 1-4/mo C3: 1-2/wk C4: 3-7/wk C5: $> 1$ /d	<b>High TG:</b> C1(ref): 130 C2: 81 C3: 92 C4: 109 C5: 45	<b>Model:</b> age, sex, total energy, education, baseline for lipid trait, current smoking status, diabetes status, physical activity index, use of LDL-lowering medication, alcohol,	<b>Subjects categorised according to their cumulative mean intake</b>  <b>HR (95% CI)</b>  <b>High TG:</b> C1(ref): 1 C2: 1.03 (0.77, 1.37)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure n Exposure assessment method	Incident cases	Model covariates	Results
	Up to 23 y (Mean 12.5 y)  Public funding	<b>Excluded:</b> prevalent dyslipidemia at baseline, use of LDL-C-lowering medications (for lipid outcomes that include LDL-C concentrations), or lack of follow-up data, implausible energy intake, incomplete FFQ  <b>n per outcome</b> <b>TG:</b> n = 2,116 <b>LDL-c:</b> n = 2,161 <b>HDL-c:</b> n = 1,703  <b>Sex:</b> 53.1% females <b>Ethnicity:</b> Caucasian <b>Age (mean±SD):</b> 54.8 ± 9.8 y	cholesterol-lowering medication. <b>Low HDL-c:</b> <40 mg/dL in men or <50 mg/dL in women.	<b>Person-years for TG   LDL-c   HDL-c per category:</b> <u>C1:</u> 8,713 7,665 7,487 <u>C2:</u> 5,336 4,852 4,531 <u>C3:</u> 5,717 5,172 4,662 <u>C4:</u> 5,984 5,615 4,760 <u>C5:</u> 2,019 2,138 1,447  <b>Servings/day Geometric mean (IQR)</b> <u>First examination:</u> 0.09 (0.49) <u>Second examination:</u> 0.09 (0.44) <u>Third examination:</u> 0.08 (0.42) <u>Fourth examination:</u> 0.05 (0.20)  <b>Serving size</b> = 355 ml  <b>Method:</b> SFFQ	<b>High LDL-c:</b> <u>C1(ref):</u> 288 <u>C2:</u> 189 <u>C3:</u> 180 <u>C4:</u> 223 <u>C5:</u> 81  <b>Low HDL-c:</b> <u>C1(ref):</u> 95 <u>C2:</u> 55 <u>C3:</u> 63 <u>C4:</u> 76 <u>C5:</u> 30	servings per day of vegetables, whole fruits, whole grains, nuts/seeds, and seafood, as well as percent energy from saturated fat, change in WC and adjustment for LCSB, and 100% fruit juice	<u>C3:</u> 1.10 (0.83, 1.46) <u>C4:</u> 1.25 (0.94, 1.68) <u>C5:</u> 1.52 (1.03, 2.25) <b>P per trend = 0.03</b>  <b>High LDL-c:</b> <u>C1(ref):</u> 1 <u>C2:</u> 1.01 (0.84, 1.22) <u>C3:</u> 0.92 (0.75, 1.11) <u>C4:</u> 1.05 (0.87, 1.28) <u>C5:</u> 1.11 (0.84, 1.47) <b>P per trend = 0.61</b>  <b>Low HDL-c</b> <u>C1(ref):</u> 1 <u>C2:</u> 0.91 (0.65, 1.27) <u>C3:</u> 1.03 (0.74, 1.44) <u>C4:</u> 1.17 (0.84, 1.63) <u>C5:</u> 1.57 (0.97, 2.54) <b>P per trend = 0.09</b>  <i>Similar results were observed when "recent intake" was used for analysis. (Recent intake being regarded as the intake one examination before development of dyslipidemia)</i>
2	<b>CARDIA</b>  USA  Duffey et al. (2010)  20 y  Mixed funding	<b>N</b> = 5,115  <b>Population sampled:</b> general population of 4 centres selected to balance subgroups of race, sex, education and age  <b>Excluded:</b> prevalent outcome at years 0 or 7, individuals who fasted for <8 h, pregnancy at time of interview, presence of diabetes, implausible energy intakes, and missing data for	<b>Fasting blood samples</b> drawn for analysis of blood lipids  <b>High TG:</b> ≥ 1.7 mmol/l or use of cholesterol-lowering medication. <b>High LDL-c:</b> ≥ 4.1 mmol/l or use of cholesterol-lowering medication. <b>Low HDL-c:</b> <1.04 mmol/l for men, <1.3 mmol/l for women or	<b>Kcal/day (mean ± SE)</b>  Year 0: 167±3 (n=5,034)  Year 7: 196±8 (n=3,877)  <b>Average of intake at 0 and 7 years used for the analysis = NR</b>  <b>Method:</b> SFFQ	<b>High TG:</b> 542  <b>High LDL-c:</b> 94  <b>Low HDL-c:</b> 252	<b>Model:</b> race, gender, centre, age, weight, smoking status, energy from food, total physical activity, energy from other beverages (low-fat milk, whole-fat milk and SSBs), and energy from alcohol	<b>Per 100kcal/d (or 250ml/d) increase</b>  <b>RR (95% CI)</b> <b>High TG:</b> 1.03 (0.99, 1.08) <b>High LDL-c:</b> 1.16 (1.08, 1.23), <b>p</b> < 0.05 <b>Low HDL-c:</b> 1.08 (1.02, 1.14), <b>p</b> < 0.05

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure n Exposure assessment method	Incident cases	Model covariates	Results
		covariates used in the analyses.  <b>n per outcome</b> <b>TG:</b> n = 2,627 <b>LDL-c:</b> n = 2,640 <b>HDL-c:</b> n = 1837  <b>Sex:</b> 53.5% females <b>Ethnicity:</b> Caucasian: 52.6%; Black:47.4% <b>Age:</b> 18 – 30 y	use of cholesterol-lowering medication.				
2	<b>Framingham -3Gen<sup>†</sup></b>  USA  Haslam et al. (2020)  Up to 9 y (Mean 6.1 y)  Public funding	<b>N</b> = 4,095  <b>Population sampled:</b> a third generation of participants of the original cohort of the Framingham Heart Study  <b>Excluded:</b> prevalent dyslipidemia at baseline, use of LDL-C-lowering medications (for lipid outcomes that include LDL-C concentrations), or lack of follow-up data, implausible energy intake, incomplete FFQ  <b>n per outcome</b> <b>TG:</b> n = 2,426 <b>LDL-c:</b> n = 2,377 <b>HDL-c:</b> n = 2,084  <b>Sex:</b> 54.3% females <b>Ethnicity:</b> Caucasian <b>Age (mean±SD):</b> 40.3 ± 8.8 y	<b>Fasting blood samples</b> drawn for analysis of blood lipids  <b>High TG:</b> ≥175 mg/dL <b>High LDL-c:</b> ≥160 mg/dL or use of LDL cholesterol-lowering medication. <b>Low HDL-c:</b> <40 mg/dL in men or <50 mg/dL in women.	<b>Range (servings/time)</b> <u>C1(ref):</u> <1/mo <u>C2:</u> 1-4/mo <u>C3:</u> 1-2/wk <u>C4:</u> 3-7/wk <u>C5:</u> >1/d  <b>Person-years for TG   LDL-c   HDL-c per category:</b> <u>C1:</u> 4,394 4,261 3,851 <u>C2:</u> 3,690 3,613 3,316 <u>C3:</u> 1,806 1,764 1,555 <u>C4:</u> 3,090 3,046 2,548 <u>C5:</u> 1,843 1,872 1,461  <b>Servings/day Geometric mean (IQR)</b> 0.12 (0.56)  <b>Serving size</b> = 355 ml  <b>Method:</b> SFFQ	<b>High TG:</b> <u>C1(ref):</u> 48 <u>C2:</u> 35 <u>C3:</u> 21 <u>C4:</u> 40 <u>C5:</u> 32  <b>High LDL-c:</b> <u>C1(ref):</u> 81 <u>C2:</u> 47 <u>C3:</u> 30 <u>C4:</u> 56 <u>C5:</u> 30  <b>Low HDL-c:</b> <u>C1(ref):</u> 25 <u>C2:</u> 27 <u>C3:</u> 15 <u>C4:</u> 28 <u>C5:</u> 14	<b>Model:</b> age, sex, total energy, education, baseline for lipid trait, current smoking status, diabetes status, physical activity index, use of LDL-lowering medication, alcohol, servings per day of vegetables, whole fruits, whole grains, nuts/seeds, and seafood, as well as percent energy from saturated fat, change in WC and adjustment for LCSB, and 100% fruit juice	<b>HR (95% CI)</b>  <b>High TG:</b> <u>C1(ref):</u> 1 <u>C2:</u> 0.89 (0.56, 1.43) <u>C3:</u> 0.92 (0.53, 1.62) <u>C4:</u> 1.04 (0.63, 1.72) <u>C5:</u> 1.49 (0.83, 2.69) <b>P per trend = 0.30</b>  <b>High LDL-c:</b> <u>C1(ref):</u> 1 <u>C2:</u> 0.76 (0.53, 1.09) <u>C3:</u> 1.15 (0.72, 1.83) <u>C4:</u> 1.18 (0.80, 1.73) <u>C5:</u> 1.04 (0.61, 1.76) <b>P per trend = 0.32</b>  <b>Low HDL-c</b> <u>C1(ref):</u> 1 <u>C2:</u> 1.15 (0.64, 2.05) <u>C3:</u> 1.15 (0.54, 2.46) <u>C4:</u> 1.55 (0.81, 2.95) <u>C5:</u> 1.07 (0.42, 2.72) <b>P per trend = 0.44</b>
<b>Exposure: SSSD+SSFD+TFJ</b>							

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure n Exposure assessment method	Incident cases	Model covariates	Results	
2	TLGS  Iran  Mirmiran et al. (2015)  3.6 y (mean)  Public funding	N = 15,005  <b>Population sampled:</b> general population from one district of Tehran  <b>Excluded:</b> those with incomplete dietary intakes or missing measures of MetS components, energy intakes (kcal/day) to energy requirements ratios beyond $\pm 3SD$ range, <b>prevalent outcome at baseline.</b>  <b>n per outcome</b> <b>TG: n = 347</b> <b>HDL-c: n = 290</b>  <b>Follow-up rate:</b> 86 %  <b>Sex:</b> 68% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 6 – 18 y	<b>Fasting blood samples</b> drawn for analysis of blood lipids in a central laboratory.  In children and adolescents: <b>High TGs:</b> $\geq 110$ mg/dl <b>Low HDL-c:</b> $< 40$ mg/dl  In those aged $> 18$ y at follow-up:  <b>High TGs:</b> $\geq 150$ mg/dl or drug treatment. <b>Low HDL-c:</b> $< 50$ mg/dl for women and $< 40$ mg/dl for men or drug treatment	ml/d (median) <u>Q1:</u> 9.3 <u>Q2:</u> 32 <u>Q3:</u> 58.6 <u>Q4:</u> 142.2  n per category for intake NR for either endpoint  Method: SFFQ	NR	<b>Model 1:</b> baseline age, sex, total energy intake, physical activity, and family history of diabetes  <b>Model 2:</b> Model 1 + dietary fibre, tea and coffee, red and processed meat, fruit, and vegetable  <b>Model 3:</b> Model 2 + BMI	<b>High TG</b>  <b>OR (95% CI)</b> <b>Model 1:</b> <u>Q1:</u> 1.00 <u>Q2:</u> 0.76 (0.24, 2.38) <u>Q3:</u> 1.57 (0.57, 4.33) <u>Q4:</u> 1.70 (0.58, 4.99) <b>P for trend = 0.156</b>  <b>Model 2:</b> <u>Q1:</u> 1.00 <u>Q2:</u> 0.74 (0.23, 2.33) <u>Q3:</u> 1.53 (0.55, 4.29) <u>Q4:</u> 1.66 (0.55, 5.05) <b>P for trend = 0.173</b>  <b>Model 3:</b> <u>Q1:</u> 1.00 <u>Q2:</u> 0.82 (0.26, 2.61) <u>Q3:</u> 1.62 (0.57, 4.58) <u>Q4:</u> 1.80 (0.59, 5.25) <b>P for trend = 0.148</b>	<b>Low HDL-c</b>  <b>OR (95% CI)</b> <b>Model 1:</b> <u>Q1:</u> 1.00 <u>Q2:</u> 0.72 (0.24, 2.16) <u>Q3:</u> 0.96 (0.33, 2.82) <u>Q4:</u> 0.55 (0.17, 1.81) <b>P for trend = 0.434</b>  <b>Model 2:</b> <u>Q1:</u> 1.00 <u>Q2:</u> 0.61 (0.19, 1.89) <u>Q3:</u> 0.93 (0.31, 2.78) <u>Q4:</u> 0.42 (0.11, 1.55) <b>P for trend = 0.320</b>  <b>Model 3:</b> <u>Q1:</u> 1.00 <u>Q2:</u> 0.65 (0.21, 2.07) <u>Q3:</u> 0.97 (0.32, 2.93) <u>Q4:</u> 0.45 (0.12, 1.66) <b>P for trend = 0.386</b>
<b>Exposure: 100% FJ</b>								
2	CARDIA  USA	Study population and exclusion criteria as for SSSD+SSFD	Same ascertainment of outcome as for SSSD+SSFD	Kcal/day (mean $\pm$ SE)  Year 0; n=5,034 115 $\pm$ 2	High TG: 542  High LDL-c: 94	<b>Model:</b> race, gender, centre, age, weight, smoking status, energy from food, total physical activity, energy from	Per 100kcal/d (or 250ml/d) increase  <b>RR (95% CI)</b> <b>High TG:</b> 0.96 (0.89, 1.04) <b>High LDL-c:</b> 1.03 (0.83, 1.27)	

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure n Exposure assessment method	Incident cases	Model covariates	Results
	Duffey et al. (2010)  20 y  Mixed funding			Year 7; n= 3,877 114±9  <b>Average of intake at 0 and 7 years used for the analysis = NR</b>  <b>Method: SFFQ</b>	<b>Low HDL-c:</b> 252	other beverages (low-fat milk, whole-fat milk and SSBs), and energy from alcohol	<b>Low HDL-c:</b> 0.98 (0.88, 1.09)

BMI, body mass index; CI, confidence interval; cm, centimetre; CVD, cardiovascular disease; d, day; FFQ, food frequency questionnaire; FJ, fruit juice; HDL-c, high-density lipoprotein cholesterol; HR, hazard ratio; IQR, interquartile range; kcal, kilocalories; kg, kilograms; kj, kilojoules; LDL-c, low density lipoprotein cholesterol; MJ, megajoules; mo, months; n, participants analysed; N, participants included in the cohort; NR, not reported; OR, odds ratio; PUFA, polyunsaturated fatty acids; RR, relative risk; SD, standard deviation; SE, standard error; SFFQ, semiquantitative food frequency questionnaire; SSB, sugar-sweetened beverages; SSFD, sugar-sweetened fruit drinks; SSFJ, sugar-sweetened fruit juices; SSSD, sugar-sweetened soft drinks; T-c, total cholesterol; TFJ, total fruit juice; TG, triglycerides; UK, United Kingdom; USA, United States of America; y, years. \*Data provided by authors. † Exposure adjusted for total energy intake using the nutrient residuals model ‡ Study identified through an update of the literature search. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Continuous measures of blood pressure

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
<b>Exposure: Total Sugars</b>							
<b>1</b>	<b>SCES</b>  Australia  Gopinath et al. (2012) <sup>36</sup>  5 y  Mixed funding	<b>N = 2,353</b>  <b>Population sampled:</b> schoolchildren from Sydney  <b>Excluded:</b> missing covariates  <b>n= 509</b> Females: 278 Males: 231  <b>Ethnicity:</b> 57% Caucasian, 19.6% East Asian, 6.8% Middle Eastern, 16.7% other  <b>Age:</b> 12 y	<b>SBP and DBP</b>  <b>BP</b> was measured after 5 minutes of resting in a seated position using an automated professional sphygmomanometer with appropriate cuff size. Three separate BP measurements were taken and averaged for analysis.	<b>g/d (mean ± SD)</b> 132.1 ± 29.4  <b>Method:</b> SFFQ	<b>Changes in total sugars</b> intake vs concurrent changes in SBP and DBP over the 5-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> crude  <b>Model 2:</b> age, ethnicity, parental education, parental history of hypertension, energy intake (residual method), baseline BP, change in height, change in body mass index, screen viewing time, and time spent in physical activity.	<b>Significant positive</b> association between changes in total sugars intake and changes in SBP and DBP in females over the 5-y follow-up. Non-significant (positive) associations for SBP and DBP in males.  <b>Per SD (51.7 g/d) increase β coefficients (SE)</b> <b>Females</b> <b>SBP (mmHg)</b> Model 1: 3.33 (1.35) <b>P = 0.01</b> Model 2: 2.28 (1.17) <b>P = 0.05</b> <b>DBP (mmHg)</b> Model 1: 2.43 (1.07) <b>P = 0.02</b> Model 2: 2.15 (0.66) <b>P = 0.001</b>  <b>Males</b> <b>SBP (mmHg)</b> Model 1: NR Model 2: 0.75 (0.84) <b>P = 0.38</b> <b>DBP (mmHg)</b> Model 1: NR Model 2: 0.89 (0.66) <b>P = 0.18</b>
<b>Exposure: Added sugars</b>							
<b>1</b>	<b>SCES</b>  Australia  Gopinath et al. (2012) <sup>35</sup>  5 y	<b>Study population and exclusion criteria as for total sugars</b>	<b>Same ascertainment of outcome as for total sugars</b>	<b>Baseline intake NR</b>  <b>Method:</b> SFFQ	<b>Changes in added sugars intake</b> vs concurrent changes in SBP and DBP over the 5-y follow-up	<b>Model 1:</b> crude  <b>Model 2:</b> age, ethnicity, parental education, parental history of hypertension, energy intake (residual method), baseline BP, change in height,	<b>Significant positive</b> association between changes in added sugars intake and changes in DBP in females over the 5-y follow-up. For changes in SBP, the association was non-significant (positive). Non-significant negative association for SBP and positive for DBP in males.  <b>Per SD (27.63 g/d) increase</b>

<sup>36</sup> Reported no observed association between change in intake of SSSD and concurrent change in BP, however, data not shown.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
	Mixed funding				<b>Data collection:</b> baseline and end of follow-up	change in body mass index, screen viewing time, and time spent in physical activity.	<b>β coefficients (SE)</b> <b>Females</b> <b>SBP (mmHg)</b> Model 1: 1.47 (1.04) <b>P = 0.16</b> Model 2: 1.24 (0.73) <b>P = 0.09</b> <b>DBP (mmHg)</b> Model 1: 1.73 (0.82) <b>P = 0.04</b> Model 2: 1.31 (0.57) <b>P = 0.02</b>  <b>Males</b> <b>SBP (mmHg)</b> Model 1: NR Model 2: -0.46 (0.93) <b>P = 0.62</b> <b>DBP (mmHg)</b> Model 1: NR Model 2: 0.18 (0.57) <b>P = 0.76</b>
<b>Exposure: Sucrose</b>							
<b>3</b>	<b>NSHDS</b>  Sweden  Winkvist et al. (2017)  10 y  Mixed funding	<b>N</b> = 40,066  <b>Population sampled:</b> General population  <b>Excluded:</b> Between visits interval <9y or >11y; >10% of the FFQ missing or missing portion sizes; implausible energy intakes, missing body weight; weight < 35 kg, length <130 cm or BMI <15 kg/m <sup>2</sup> .  <b>n</b> = 15,995 Females = 8354 Males = 7,641  <b>Ethnicity:</b> Caucasian <b>Age:</b> 30 – 60 y	<b>SBP</b>  <b>Blood pressure</b> was measured once, after 5 min rest and in supine position, using a sphygmomanometer.	<b>E% (mean ± SD)</b> Females: 6.5 ± 2.6 Males: 6.6 ± 2.9  <b>g/d (mean ± SD)</b> Females: 24.4 ± 12.6 Males: 32.2 ± 18.3  <b>Method:</b> SFFQ	<b>Changes in sucrose</b> intake vs changes in SBP over the 10-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> baseline SBP, year of study participation, age, education, smoking status and physical activity at the beginning of the period  <i>Joint model i.e. whole grain, PUFA, cholesterol, trans-fatty acids and sucrose entered in the same model</i>	Non-significant negative association in females and positive association in males between changes in sucrose intake and changes in SBP over the 10-y follow-up.  <b>Per each 1E% increase in intake β ± SE</b> <b>Females:</b> -0.66 ± 0.38, <b>P=0.08</b> <b>Males:</b> 0.38 ± 0.32, <b>P=0.22</b>  <b>No results reported for DBP</b>
<b>Exposure: Fructose</b>							

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
1	<b>SCES</b>  Australia  Gopinath et al. (2012)  5 y  Mixed funding	<b>Study population and exclusion criteria as for total sugars</b>	<b>Same ascertainment of outcome as for total sugars</b>	<b>Baseline intake NR</b>  <b>Method:</b> SFFQ	<b>Changes in fructose intake</b> vs concurrent changes in SBP and DBP over the 5-y follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> crude  <b>Model 2:</b> age, ethnicity, parental education, parental history of hypertension, energy intake (residual method), baseline BP, change in height, change in body mass index, screen viewing time, and time spent in physical activity.	<b>Significant positive</b> association between changes in fructose intake and changes in SBP and DBP in females over the 5-y follow-up. Non-significant (positive) associations for SBP and DBP in males.  <b>Per SD (14.19 g/d) increase <math>\beta</math> coefficients (SE)</b>  <b>Females</b> SBP (mmHg) Model 1: 2.29 (0.97) <b>P = 0.02</b> Model 2: 1.80 (0.82) <b>P = 0.03</b> DBP (mmHg) Model 1: 1.54 (0.77) <b>P = 0.05</b> Model 2: 1.67 (0.61) <b>P = 0.01</b>  <b>Males</b> SBP (mmHg) Model 1: NR Model 2: 0.81 (0.73) <b>P = 0.27</b> DBP (mmHg) Model 1: NR Model 2: 0.34 (0.60) <b>P = 0.57</b>
3	<b>TLGS</b>  Iran  Bahadoran et al. (2017)  6.7 y (mean)  Public funding	<b>N = 15,005</b>  <b>Population sampled:</b> general population from one district of Tehran  <b>Excluded:</b> Unusual energy intake (<800 kcal/day or >4200 kcal/day), or were on specific diets for hypertension, diabetes or dyslipidemia; those with a history of CVD at baseline.  <b>n = 2,369</b>	<b>SBP and DBP</b>  <b>Blood pressure</b> was measured after a 15-min rest in the sitting position. Two measurements of blood pressure were taken on the right arm using a standardized mercury sphygmomanometer; the mean of the two measurements was considered to be the blood pressure of the participant.	<b>%E (mean <math>\pm</math> SD)</b> 6.4 $\pm$ 3.7  <b>Method:</b> SFFQ	<b>Fructose</b> intake at baseline vs changes in TG and HDL-c over the follow-up  <b>Data collection:</b> baseline and end of follow-up	<b>Model:</b> age	<b>Significant positive</b> associations between baseline fructose intake and changes in SBP and DBP over the follow-up.  <b>Per each 1 %E increase <math>\beta</math> coefficients (95% CI)</b> SBP: 0.217 (0.063, 0.371) DBP: 0.267 (0.157, 0.376)



RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results
		<b>Follow-up rate:</b> 99.5% <b>Sex:</b> 56.5% females <b>Ethnicity:</b> Caucasian <b>Age (mean ± SD):</b> 3 19 y					
<b>Exposure: SSSD+SSFD+SSFJ</b>							
<b>1</b>	<b>WAPCS</b>  Australia  Ambrosini et al. (2013)  3 y  Unclear	<b>N</b> = 2,868  <b>Population sampled:</b> offspring from mothers from the Raine study <b>Excluded:</b> Subjects who reported not fasting before venepuncture.  <b>n</b> = 1,366 females= 660 males= 706  <b>Follow-up rate:</b> 94%  <b>Sex:</b> 48.3% females  <b>Ethnicity:</b> Caucasian  <b>Age (mean ± SD):</b> 14.0 ± 0.2 y	<b>SBP and DBP</b> Blood pressure was measured by using an oscillometric sphygmomanometer after subjects rested supine for 5 min. Measurements were recorded every 2 min for 10 min; average values, with the exclusion of the first measurement, were used for analyses.	<b>g/d (range (mean ± SD))</b> <b>T1:</b> 0 – 130 (48 ± 39) <b>T2:</b> 130 – 329 (223 ± 59) <b>T3:</b> 331 – 2,876 (665 ± 351)  <b>n for those changing tertiles of SSB intake = NR</b>  <b>Method:</b> SFFQ	<b>Changes in SSSD+SSFD+S SFJ intake vs percent of change in SBP and DBP over the 3-y follow-up</b>  <b>Data collection:</b> baseline and end of follow-up	<b>Model 1:</b> age, pubertal stage, physical fitness, dietary misreporting, maternal education, and family income  <b>Model 2:</b> Model 1 + BMI  <b>Model 3:</b> Model 2 + Healthy and Western dietary pattern scores	Non-significant associations between changes in SSSD+SSFD+SSFJ intake and changes in SBP (positive) and DBP (negative) over the 3-y follow-up.  <b>Per each tertile of intake increase Δ% (95% CI) vs T1</b>  <b>Females - SBP</b> <b>T1 (ref):</b> 0 <b>Model 1:</b> <b>T2:</b> 0.2 (-1.1, 1.5) <b>T3:</b> 1.7 (0.3, 3.1) <b>P for trend = 0.02</b>  <b>Model 2:</b> <b>T2:</b> 0.2 (-1.1, 1.5) <b>T3:</b> 0.9 (-0.5, 2.3) <b>P for trend = 0.24</b>  <b>Model 3:</b> <b>T2:</b> 0.1 (-1.2, 1.4) <b>T3:</b> 0.8 (-0.8, 2.4) <b>P for trend = 0.36</b>  <b>Females - DBP</b> <b>T1 (ref):</b> 0 <b>Model 1:</b> <b>T2:</b> -0.6 (-2.4, 1.2)
							<b>Per each tertile of intake increase Δ% (95% CI) vs T1</b>  <b>Males - SBP</b> <b>T1 (ref):</b> 0 <b>Model 1:</b> <b>T2:</b> 0.5 (-0.9, 1.8) <b>T3:</b> 0.7 (-0.7, 2.1) <b>P for trend = 0.34</b>  <b>Model 2:</b> <b>T2:</b> 0.1 (-1.2, 1.5) <b>T3:</b> 0.3 (-1.1, 1.6) <b>P for trend = 0.69</b>  <b>Model 3:</b> <b>T2:</b> 0.3 (-1.0, 1.7) <b>T3:</b> 0.8 (-0.7, 2.2) <b>P for trend = 0.29</b>  <b>Males - DBP</b> <b>T1 (ref):</b> 0 <b>Model 1:</b> <b>T2:</b> 0.2 (-1.6, 2.1)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original Cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcomes	Exposure and method	Exposure-outcome relationships	Model covariates	Results	
							<u>T3</u> : -0.8 (-2.7, 1.1) <b>P for trend = 0.40</b>  <b>Model 2:</b> <u>T2</u> : -0.6 (-2.4, 1.2) <u>T3</u> : -1.1 (-3, 0.9) <b>P for trend = 0.28</b>  <b>Model 3:</b> <u>T2</u> : -0.8 (-2.7, 1) <u>T3</u> : -1.8 (-4, 0.4) <b>P for trend = 0.12</b>	<u>T3</u> : -0.4 (-2.2, 1.5) <b>P for trend = 0.67</b>  <b>Model 2:</b> <u>T2</u> : 0.1 (-1.8, 1.9) <u>T3</u> : -0.6 (-2.4, 1.3) <b>P for trend = 0.53</b>  <b>Model 3:</b> <u>T2</u> : 0.5 (-1.4, 2.4) <u>T3</u> : -0.2 (-2.2, 1.8) <b>P for trend = 0.84</b>

BMI, body mass index; BP, blood pressure; CI, confidence interval; cm, centimetre; CVD, cardiovascular disease; d, day; DBP, diastolic blood pressure; FFQ, food frequency questionnaire; kcal, kilocalories; n, participants analysed; N, participants included in the cohort; NR, not reported; PUFA, polyunsaturated fatty acids; SBP, systolic blood pressure; SD, standard deviation; SE, standard error; SFFQ, semiquantitative food frequency questionnaire; SSFD, sugar-sweetened fruit drinks; SSFJ, sugar-sweetened fruit juices; SSSD, sugar-sweetened soft drinks; y, years. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Incidence of hypertension

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: total fructose</b>							
<b>1</b>	<b>HPFS</b>  USA  Forman et al. (2009)  18 y  Public funding	<b>N</b> = 51,529  <b>Population sampled:</b> male health professionals  <b>Excluded:</b> prevalent HTN at baseline  <b>n</b> = 37,375  <b>Sex:</b> males <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 40-75 y	Self-reported HTN in any follow-up questionnaire (every 2 years from baseline).  Positive predictive value of incident HTN: 100%, as assessed in a validation study against medical record review (SBP and DBP >140 and >90 mmHg, respectively) in a subset of men.  Negative predictive value when HTN is not reported = NR	<b>E% (median (range))</b> <b>Q1 (ref):</b> 5.7 (0.5-6.9) <b>Q2:</b> 7.8 (7.0-8.6) <b>Q3:</b> 9.3 (8.7-10.1) <b>Q4:</b> 11.0 (10.2-12.1) <b>Q5:</b> 13.9 (12.2-36.2)  <b>Person years:</b> <b>Q1 (ref):</b> 84,933 <b>Q2:</b> 85,452 <b>Q3:</b> 85,387 <b>Q4:</b> 85,023 <b>Q5:</b> 85,268  <b>Exposure assessment:</b> SFFQ	<b>Q1 (ref):</b> 2,461 <b>Q2:</b> 2,213 <b>Q3:</b> 2,123 <b>Q4:</b> 2,195 <b>Q5:</b> 2,200	<b>Model 1:</b> age and BMI  <b>Model 2:</b> model 1 + physical activity, smoking status, family history of hypertension, intakes of alcohol, caffeine, folate, and vitamin C, and total energy intake	<b>Model 1; RR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 0.89 (0.84, 0.95) <b>Q3:</b> 0.85 (0.80, 0.91) <b>Q4:</b> 0.88 (0.83, 0.94) <b>Q5:</b> 0.89 (0.84, 0.95)  <b>Model 2; RR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 0.95 (0.89, 1.00) <b>Q3:</b> 0.93 (0.87, 0.98) <b>Q4:</b> 0.97 (0.91, 1.03) <b>Q5:</b> 0.99 (0.93, 1.05)
<b>1</b>	<b>NHS</b>  USA  Forman et al. (2009)  20 y  Public funding	<b>N</b> = 121,770  <b>Population sampled:</b> female nurses  <b>Excluded:</b> prevalent HTN at baseline  <b>n</b> = 88,540  <b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~93%+) <b>Age:</b> 30-55 y	Self-reported HTN in any follow-up questionnaire (every 2 years from baseline).  Positive predictive value of incident HTN: 100%, as assessed in a validation study against medical record review (SBP and DBP >140 and >90 mmHg, respectively) in a subset of women.  Negative predictive value when HTN is not reported = NR	<b>E% (median (range))</b> <b>Q1 (ref):</b> 6.0 (0.1-7.2) <b>Q2:</b> 8.1 (7.3-8.9) <b>Q3:</b> 9.7 (9.0-10.5) <b>Q4:</b> 11.4 (10.6-12.6) <b>Q5:</b> 14.3 (12.7-37.8)  <b>Person years:</b> <b>Q1 (ref):</b> 186,935 <b>Q2:</b> 204,417 <b>Q3:</b> 208,345 <b>Q4:</b> 206,060 <b>Q5:</b> 184,889  <b>Exposure assessment:</b> SFFQ	<b>Q1 (ref):</b> 6,055 <b>Q2:</b> 6,427 <b>Q3:</b> 6,269 <b>Q4:</b> 6,309 <b>Q5:</b> 6,047	<b>Model 1:</b> age and BMI  <b>Model 2:</b> model 1 + physical activity, smoking status, family history of hypertension, intakes of alcohol, caffeine, folate, and vitamin C, and total energy intake	<b>Model 1; RR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 0.95 (0.92, 0.98) <b>Q3:</b> 0.90 (0.87, 0.93) <b>Q4:</b> 0.92 (0.89, 0.95) <b>Q5:</b> 0.97 (0.94, 1.00)  <b>Model 2; RR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 0.98 (0.94, 1.01) <b>Q3:</b> 0.94 (0.90, 0.97) <b>Q4:</b> 0.96 (0.92, 0.99) <b>Q5:</b> 1.02 (0.99, 1.06)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
1	<b>NHS II</b>  USA  Forman et al. (2009)  14 y  Public funding	<b>N</b> = 116,671  <b>Population sampled:</b> female nurses  <b>Excluded:</b> prevalent HTN at baseline  <b>n</b> = 97,315  <b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 25-42 y	Self-reported HTN in any follow-up questionnaire (every 2 years from baseline).  Positive predictive value of incident HTN: 100%, as assessed in a validation study against medical record review (SBP and DBP >140 and >90 mmHg, respectively) in a subset of women.  Negative predictive value when HTN is not reported = NR	<b>E% (median (range))</b> <b>Q1 (ref):</b> 5.7 (0.7-6.7) <b>Q2:</b> 7.6 (6.8-8.3) <b>Q3:</b> 9.1 (8.4-9.9) <b>Q4:</b> 10.9 (10.0-12.1) <b>Q5:</b> 14.3 (12.2-45.9)  <b>Person-years:</b> <b>Q1 (ref):</b> 215,222 <b>Q2:</b> 217,250 <b>Q3:</b> 217,887 <b>Q4:</b> 218,294 <b>Q5:</b> 216,995  <b>Exposure assessment:</b> SFFQ	<b>Q1 (ref):</b> 3,600 <b>Q2:</b> 3,250 <b>Q3:</b> 3,074 <b>Q4:</b> 2,816 <b>Q5:</b> 3,123	<b>Model 1:</b> age and BMI  <b>Model 2:</b> model 1 + physical activity, smoking status, family history of hypertension, intakes of alcohol, caffeine, folate, and vitamin C, and total energy intake	<b>Model 1; RR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 0.96 (0.91, 1.00) <b>Q3:</b> 0.96 (0.91, 1.00) <b>Q4:</b> 0.92 (0.87, 0.97) <b>Q5:</b> 1.03 (0.98, 1.08)  <b>Model 2; RR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 0.98 (0.93, 1.03) <b>Q3:</b> 0.98 (0.93, 1.03) <b>Q4:</b> 0.94 (0.89, 0.99) <b>Q5:</b> 1.03 (0.98, 1.08)
<b>Exposure: SSSD</b>							
2	<b>KoGES</b>  South Korea  Kwak et al. (2018)  8 y (mean)  Public funding	<b>N</b> = 10,030  <b>Population sampled:</b> general population living in Ansan (urban) and Ansong (rural) areas  <b>Excluded:</b> history of HTN, diabetes, CVD and cancer.  <b>n</b> = 5,775  <b>Sex:</b> 54.4% females <b>Ethnicity:</b> Asian <b>Age:</b> >30 years	Subjects diagnosed with HTN, taking blood pressure medicines or with SBP >140 or DBP >90 mmHg at follow-up check-ups (every 2 years) were considered incident cases of HTN.	<b>Servings/week (mean, median (range))</b> <b>Q1 (ref):</b> 0 <b>Q2:</b> 0.29, 0.23 (0.12-0.52) <b>Q3:</b> 1.03, 0.83 (0.57-1.62) <b>Q4:</b> 4.38, 3.50 (1.73-42.00)  <b>Serving size</b> = 200 mL  <b>n/person-years:</b> <b>Q1 (ref):</b> 1,525/7,468 <b>Q2:</b> 1,154/5,818 <b>Q3:</b> 1,430/6,985 <b>Q4:</b> 1,489/7,157  <b>Exposure assessment:</b> SFFQ	<b>Q1 (ref):</b> 331 <b>Q2:</b> 245 <b>Q3:</b> 295 <b>Q4:</b> 304	<b>Model 1:</b> age, sex and total energy intake  <b>Model 2:</b> model 1 + education, income status, physical activity, alcohol use and cigarette smoking  <b>Model 3:</b> model 2 + intake frequencies of whole grains, dairy, fish and sodium and potassium  <b>Adjustments as specified in Model 2 did not materially change the RRs as estimated in Model 3 (not shown)</b>	<b>Model 1; HR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 1.012 (0.858, 1.194) <b>Q3:</b> 1.065 (0.909, 1.248) <b>Q4:</b> 1.139 (0.967, 1.341) <b>P per trend=0.106</b>  <b>Model 3; HR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 1.039 (0.872, 1.236) <b>Q3:</b> 1.122 (0.949, 1.325) <b>Q4:</b> 1.214 (1.019, 1.445) <b>P per trend=0.033</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: SSSD+SSFD</b>							
<b>1</b>	<b>CARDIA</b>  USA  Duffey et al. (2010)  20 y  Mixed funding	<b>N</b> = 5,115  <b>Population sampled:</b> general population of 4 centres selected to balance subgroups of race, sex, education and age  <b>Excluded:</b> pregnancy, fasting < 8 h at any examination (baseline, 7 and 20 y); SBP ≥ 130, DBP ≥ 85 mmHg, or use of antihypertensive medication at baseline or 7-y visit  <b>Follow-up rate:</b> 61%  <b>n</b> = 2,639  <b>Sex:</b> 54.7% females <b>Ethnicity:</b> Caucasian 52.6%, Black 47.4% <b>Age:</b> 18-30 y	Incident hypertension was defined as SBP ≥130, DBP ≥85 mmHg, or use of antihypertensive medication at the 20-y visit. Seated BP was measured 3 times; the average of the last 2 measurements was used.	<b>Kcal/day (mean±SEM)</b>  <b>Year 0:</b> n=5,034 167±3  <b>Year 7:</b> n= 3,877 196±8  Exposure reported for the whole study sample (not restricted to subjects available for the analysis on HTN). Average of intake at 0 and 7 years used for the analysis = NR  <b>Exposure assessment:</b> SFFQ	609	<b>Model:</b> race, gender, centre, age, weight, smoking status, energy from food, total physical activity, energy from other beverages (low-fat milk, whole-fat milk and fruit juice), and energy from alcohol.	<b>Per 100 kcal/d increase* RR (95% CI)</b> 1.04 (1.00, 1.08)  Data from supplemental material
<b>1</b>	<b>HPFS</b>  USA  Cohen et al. (2012)  22 y  Public funding	<b>N</b> = 51,529  <b>Population sampled:</b> male health professionals  <b>Excluded:</b> prevalent HTN at baseline  <b>n</b> = 37,360	<b>Same ascertainment of outcome as for total fructose</b>	<b>Servings/time (range)</b> <b>C1 (ref):</b> <1/mo <b>C2:</b> 1-4/mo <b>C3:</b> 2-6/wk <b>C4:</b> ≥1/d  <b>Serving size</b> = 12oz (355mL)  <b>Person-years:</b>	<b>C1 (ref):</b> 5,038 <b>C2:</b> 3,198 <b>C3:</b> 3,872 <b>C4:</b> 1,331	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + race, family history of HTN, physical activity, calcium, magnesium and vitamin D intake, cereal fibre and trans-fat intake, carbohydrate consumption, DASH-style diet, total fructose consumption, total energy intake, alcohol, intent of losing weight,	<b>Model 1; HR (95% CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 0.97 (0.92, 1.01) <b>C3:</b> 1.05 (1.00, 1.09) <b>C4:</b> 1.09 (1.02, 1.16)  <b>Model 2; HR (95% CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 0.96 (0.92, 1.01) <b>C3:</b> 1.02 (0.98, 1.07) <b>C4:</b> 1.04 (0.97, 1.12)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		<b>Sex:</b> males <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 40-75 y		<u>C1 (ref):</u> 172,999 <u>C2:</u> 118,553 <u>C3:</u> 142,434 <u>C4:</u> 49,658  <b>Exposure assessment:</b> SFFQ		smoking status, non-narcotic analgesic use and ASB intake  <b>Model 3:</b> model 2 + BMI, BMI <sup>2</sup> and weight change	<b>Model 3; HR (95% CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 0.97 (0.93, 1.02) <u>C3:</u> 1.04 (1.00, 1.10) <u>C4:</u> 1.06 (0.99, 1.14)  <b>A stronger positive (significant) association was observed for ASBs HR (95% CI)</b> <u>C4 vs C1:</u> 1.20 (1.14, 1.26)
1	<b>NHS</b>  USA  Cohen et al. (2012)  28 y  Public funding	<b>N</b> = 121,770  <b>Population sampled:</b> female nurses  <b>Excluded:</b> prevalent HTN at baseline  <b>n</b> = 88,540  <b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~93%+) <b>Age:</b> 30-55 y	<b>Same ascertainment of outcome as for total fructose</b>	<b>Servings/time (range)</b> <u>C1 (ref):</u> <1/mo <u>C2:</u> 1-4/mo <u>C3:</u> 2-6/wk <u>C4:</u> ≥1/d  <b>Serving size</b> = 12oz (355mL)  <b>Person-years:</b> <u>C1 (ref):</u> 556,939 <u>C2:</u> 402,891 <u>C3:</u> 276,384 <u>C4:</u> 129,827  <b>Exposure assessment:</b> SFFQ	<u>C1 (ref):</u> 17,989 <u>C2:</u> 11,849 <u>C3:</u> 8,186 <u>C4:</u> 3,998	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + race, family history of HTN, physical activity, calcium, magnesium and vitamin D intake, cereal fibre and trans-fat intake, carbohydrate consumption, DASH-style diet, total fructose consumption, total energy intake, alcohol, intent of losing weight, smoking status, oral contraceptive use, non-narcotic analgesic use and ASB intake  <b>Model 3:</b> model 2 + BMI, BMI <sup>2</sup> and weight change	<b>Model 1; HR (95% CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 1.03 (1.00, 1.05) <u>C3:</u> 1.09 (1.06, 1.12) <u>C4:</u> 1.22 (1.18, 1.27)  <b>Model 2; HR (95% CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 1.00 (0.98, 1.03) <u>C3:</u> 1.02 (0.99, 1.05) <u>C4:</u> 1.11 (1.07, 1.15)  <b>Model 3; HR (95% CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 1.02 (0.99, 1.04) <u>C3:</u> 1.04 (1.01, 1.07) <u>C4:</u> 1.12 (1.08, 1.17)  <b>A similar positive association was observed for ASBs HR (95% CI)</b> <u>C4 vs C1:</u> 1.11 (1.08, 1.14)
1	<b>NHS II</b>  USA	<b>N</b> = 116,671  <b>Population sampled:</b> female nurses	<b>Same ascertainment of outcome as for total fructose</b>	<b>Servings/time (range)</b> <u>C1 (ref):</u> <1/mo <u>C2:</u> 1-4/mo <u>C3:</u> 2-6/wk	<u>C1 (ref):</u> 8,394 <u>C2:</u> 5,137 <u>C3:</u> 5,027 <u>C4:</u> 3,315	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + race, family history of HTN, physical activity, calcium, magnesium and vitamin D	<b>Model 1; HR (95% CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 1.02 (0.98, 1.05) <u>C3:</u> 1.14 (1.10, 1.18) <u>C4:</u> 1.39 (1.34, 1.46)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Cohen et al. (2012)  16 y  Public funding	<b>Excluded:</b> prevalent HTN at baseline  <b>n</b> = 97,991  <b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 25-42 y		<u>C4:</u> ≥1/d  <b>Serving size</b> = 12oz (355mL)  <b>Person-years:</b> <u>C1</u> (ref): 456,363 <u>C2:</u> 307,057 <u>C3:</u> 303,437 <u>C4:</u> 176,141  <b>Exposure assessment:</b> SFFQ		intake, cereal fibre and trans-fat intake, carbohydrate consumption, DASH-style diet, total fructose consumption, total energy intake, alcohol, intent of losing weight, smoking status, oral contraceptive use, non-narcotic analgesic use and ASB intake  <b>Model 3:</b> model 2 + BMI, BMI <sup>2</sup> and weight change	<b>Model 2; HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2:</u> 0.97 (0.94, 1.01) <u>C3:</u> 1.02 (0.98, 1.06) <u>C4:</u> 1.12 (1.06, 1.17)  <b>Model 3; HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2:</u> 1.00 (0.96, 1.04) <u>C3:</u> 1.07 (1.03, 1.11) <u>C4:</u> 1.17 (1.11, 1.23)  <b>A similar positive association was observed for ASBs</b> <b>HR (95% CI)</b> <u>C4 vs C1:</u> 1.11 (1.08, 1.14)
1	<b>SUN</b>  Spain  Sayon-Orea et al. (2015)  8.1 y (median)  Public funding	<b>N</b> = 21,678  <b>Population sampled:</b> University graduates, mainly health professionals  <b>Excluded:</b> prevalent HTN at baseline (medical diagnosis of HTN, SBP ≥140 mmHg, DBP ≥90 mmHg, or any use of antihypertensive medication), implausible energy intake at baseline (< 800 kcal/d for men and < 500 kcal/d for women or > 4000 kcal/d for men and > 3500 kcal/d for	Incident cases of HTN were identified by self-reporting new medical diagnosis of HTN at follow-up questionnaires (SBP ≥140 mmHg, a DBP ≥90 mmHg, or any use of antihypertensive medication).  Positive predictive value for incident HTN: 82.3%.  Negative predictive value when HTN is NR = 85.4% as assessed in a validation study by direct measurement of	<b>Servings/week (median, range)</b> <u>C1</u> (non-consumers, ref): 0 <u>C2:</u> 1 (<7/wk) <u>C3:</u> 8 (≥7/wk)  <b>Serving size</b> = 200 mL  <b>n/person-years:</b> <u>C1</u> (ref): 3,250/23,163 <u>C2:</u> 9,260/71,542 <u>C3:</u> 1,333/10,140  <b>Exposure assessment:</b> SFFQ	<u>C1</u> (ref): 374 <u>C2:</u> 798 <u>C3:</u> 136	<b>Model 1:</b> crude  <b>Model 2:</b> age and sex  <b>Model 3:</b> model 2 + baseline BMI, family history of HTN, self-reported hypercholesterolemia, physical activity, years of university education, smoking status, total energy intake, energy adjusted sodium, potassium, low fat dairy, olive oil, fruit, vegetables, cereals, legumes, meat, whole fat dairy and fish consumption  <b>Model 4:</b> model 3 + alcohol intake  <b>Adjustments as specified in Model 3 did not materially change the RRs as estimated in Model 4 (not shown)</b>	<b>Model 1; HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2:</u> 1.17 (1.03, 1.33) <u>C3:</u> 1.57 (1.28, 1.91)  <b>Model 2; HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2:</u> 1.08 (0.95, 1.22) <u>C3:</u> 1.39 (1.14, 1.70)  <b>Model 4; HR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2:</u> 1.07 (0.94, 1.22) <u>C3:</u> 1.34 (1.09, 1.65)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		women), chronic disease at baseline, lost to follow-up and missing covariate data.  <b>Follow-up rate:</b> 85.4%  <b>n</b> = 13,843 <b>Sex:</b> 63.4% women <b>Ethnicity:</b> Caucasian <b>Age (mean ±SD):</b> 36.4±10.8	blood pressure in 79 subjects reporting and 48 subjects not reporting a diagnosis of HTN <sup>37</sup> .				
<b>Exposure: SSSD+SSFD+TFJ</b>							
<b>2</b>	<b>TLGS</b>  Iran  Mirmiran et al. (2015)  3.6 y (mean)  Public funding	<b>N</b> = 15,005  <b>Population sampled:</b> general population from one district of Tehran  <b>Excluded:</b> incomplete dietary intake or missing measures of MetS components, reported energy intake to energy requirements ratio beyond ±3SD, prevalent hypertension or age <6 y or >18 y at baseline for this outcome (survey 3).  <b>Follow-up rate:</b> 86% <b>n</b> = 424 <b>Sex:</b> 68 % females <b>Ethnicity:</b> Caucasian	Blood pressure measured twice, after participants were seated for 15min, with a minimum interval of 30s; the mean of the two measurements was considered the patient's blood pressure.  Incident hypertension was defined as SBP ≥ 130mmHg, DBP ≥ 85 mmHg or antihypertensive drug treatment during follow-up (survey 4).	<b>Median intake (ml/d)</b> <u>Q1 (ref):</u> 9.3 <u>Q2:</u> 32.0 <u>Q3:</u> 58.6 <u>Q4:</u> 142.2  <b>N of subjects per quartile for this outcome NR</b>  <b>Exposure assessment:</b> SFFQ	<b>Number of incident cases NR</b>	<b>Model 1:</b> age, sex, total energy intake, physical activity and family history of diabetes  <b>Model 2:</b> model 1 + dietary fibre, tea and coffee, red a processed meat, fruit and vegetables  <b>Model 3:</b> model 2 + BMI	<b>Model 1; OR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.46 (0.45, 4.77) <u>Q3:</u> 2.66 (0.89, 7.96) <u>Q4:</u> 2.41 (0.79, 7.73) <b>P per trend = 0.070</b>  <b>Model 2; OR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.47 (0.45, 4.82) <u>Q3:</u> 2.68 (0.89, 8.11) <u>Q4:</u> 2.45 (0.78, 7.70) <b>P per trend = 0.072</b>  <b>Model 3; OR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.73 (0.52, 5.74) <u>Q3:</u> 3.02 (0.98, 9.25) <u>Q4:</u> 2.90 (0.91, 9.26) <b>P per trend = 0.043</b>

<sup>37</sup> Alonso A, Beunza JJ, Delgado-Rodríguez M, Martínez-Gonzalez MA. Validation of self-reported diagnosis of hypertension in a cohort of university graduates in Spain. BMC Public Health 2005;5:94.



RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		Age: 6-18 y					
<b>Exposure: 100% FJ</b>							
<b>1</b>	<b>CARDIA</b>  USA  Duffey et al. (2010)  20 y  Mixed funding	<b>Study population and exclusion criteria as for SSSD+SSFD</b>	<b>Same ascertainment of outcome as for SSSD+SSFD</b>	<b>Kcal/day (mean±SEM)</b>  <b>Year 0:</b> n=5,034 115±2  <b>Year 7:</b> n= 3,877 114±9  Exposure reported for the whole study sample (not restricted to subjects available for the analysis on HTN). Average of intake at 0 and 7 years used for the analysis = NR  <b>Exposure assessment:</b> SFFQ	609	<b>Model:</b> race, gender, centre, age, weight, smoking status, energy from food, total physical activity, energy from the three other beverages, and energy from alcohol.	<b>Per 100 kcal increase* HR (95% CI)</b> 0.99 (0.96, 1.03)  Data from supplemental material
<b>1</b>	<b>WHI</b>  USA  Auerbach et al. (2017)  7.8 y (mean)  Public funding	<b>N = 122,970</b>  <b>Population sampled:</b> Postmenopausal women recruited from 40 clinical centres  <b>Excluded:</b> energy intake outliers on baseline FFQ (defined as ≤600 kcal/d or ≥5000 kcal/d), baseline self-reported past or current hypertension, missing answers to the two	Self-reported incident HTN. Standardized medical history questionnaires asking about new treatment of HTN were completed every 6-12 months until the conclusion of the study.  Participants were considered to have incident HTN if they initiated medication to treat hypertension.	<b>oz/d†</b> <b>Median (range):</b> <b>Q1 (ref):</b> 0 (0) <b>Q2:</b> 1 (0.06-1.7) <b>Q3:</b> 2.6 (1.8-3.8) <b>Q4:</b> 4.9 (3.9-6.5) <b>Q5:</b> 7.8 (6.6-36.8)  1 oz ≡ 29.6 mL  <b>Person-year:</b> <b>Q1 (ref):</b> 58,299 <b>Q2:</b> 100,796 <b>Q3:</b> 100,614 <b>Q4:</b> 99,971 <b>Q5:</b> 99,467	<b>Q1 (ref):</b> 5,994 <b>Q2:</b> 10,087 <b>Q3:</b> 9,971 <b>Q4:</b> 10,036 <b>Q5:</b> 10,114	<b>Model:</b> age, education level, race/ethnicity, smoking status, physical activity, BMI, hormone replacement therapy status, study arm and total energy intake  <i>Univariate and multivariable-adjusted models yielded nearly identical HR 95%CI – Results of the univariate model NR in paper</i>	<b>HR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 0.98 (0.94, 1.01) <b>Q3:</b> 0.97 (0.94, 1.01) <b>Q4:</b> 0.98 (0.94, 1.01) <b>Q5:</b> 1.01 (0.97, 1.04) <b>P per trend=0.21</b>

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		100% fruit juice questions on the FFQ.  <b>n</b> = 80,539  <b>Sex:</b> women <b>Ethnicity:</b> ~ 84% Caucasian, 7.6% Black, Hispanic/Latino 4% and 3% Asian/Pacific <b>Age:</b> 50-79y	Positive predictive value of incident HTN: nearly 80%  Negative predictive value when HTN is not reported = nearly 100%	<b>Exposure assessment:</b> SFFQ			

ASB, artificially sweetened beverages; BMI, body mass index; BP, blood pressure; CI, confidence interval; CVD, cardiovascular disease; d, day; DASH, Dietary Approaches to Stop Hypertension; HR, hazard ratio; HRT, hormone replacement therapy; HTN, hypertension; mo, month; n, participants analysed; N, participants included in the cohort; NR, not reported; OR, odds ratio; RR, risk ratio; SBP, systolic blood pressure; SEM, standard error of the mean; SFFQ, semiquantitative food frequency questionnaire; SSFD, sugar-sweetened fruit drinks; SSSD, sugar-sweetened soft drinks; TFJ, total fruit juices; USA, United States of America; wk, week; y, years. \*Data provided by the authors † Exposure adjusted for total energy intake using the nutrient residuals model. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Cardiovascular diseases (incidence and mortality)

### Cardiovascular diseases (composite endpoint)

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: total sugars</b>							
<b>1</b>	<b>EPIC-Utrecht</b>  The Netherlands  Beulens et al. (2007)*  9 y (mean)  Public funding	<b>N</b> = 17,357 <b>Population sampled:</b> Breast cancer screening participants  <b>Excluded:</b> not consent to linkage with vital status registries, missing questionnaires, energy intake of <500 kcal/day or >6,000 kcal/day, prevalent CHD, cerebrovascular disease, or diabetes.  <b>n</b> = 15,714 <b>Sex:</b> females <b>Ethnicity:</b> Caucasian <b>Age:</b> 49-70 y	<b>CVD incidence</b> defined as fatal and non-fatal cases of CHD and stroke ( <b>ICD-9-CM 410 to 414, 427.5; ICD-9-CM 430 to 438</b> ).  Morbidity data: from the Dutch Centre for Health Care Information (standardized computerized register of hospital discharge diagnoses). Information on vital status: linkage with the municipal administration registries. Causes of death: from the women's general practitioners and coded by 2 independent physicians.	<b>g/day + Mean (SD)</b> <b>Q1</b> (ref): 75 (22) <b>Q2:</b> 100 (22) <b>Q3:</b> 116 (26) <b>Q4:</b> 140 (37)  <b>n/person years</b> <b>Q1</b> (ref): 3,928/35,278 <b>Q2:</b> 3,929/35,429 <b>Q3:</b> 3,929/35,504 <b>Q4:</b> 3,928/35,423  <b>Exposure assessment:</b> SFFQ	<b>Q1</b> (ref): 209 <b>Q2:</b> 178 <b>Q3:</b> 200 <b>Q4:</b> 212	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + hypertension, cholesterolemia, smoking, BMI, SBP, physical activity, menopausal status, HRT use, oral contraceptives use, alcohol intake, total energy intake, energy-adjusted intake of vitamin E; protein, dietary fiber, folate; saturated fat; and poly- and monounsaturated fat	<b>Model 1; HR (95% CI)</b> <b>Q1</b> (ref): 1 <b>Q2:</b> 0.77 (0.63, 0.94) <b>Q3:</b> 0.83 (0.68, 1.00) <b>Q4:</b> 0.84 (0.70, 1.02)  <b>Model 2; HR (95% CI)</b> <b>Q1</b> (ref): 1 <b>Q2:</b> 0.91 (0.73, 1.15) <b>Q3:</b> 1.00 (0.77, 1.31) <b>Q4:</b> 1.04 (0.72, 1.48)
<b>1</b>	<b>NIH-AARP</b>  USA  Tasevska et al. (2014)*  13 y	<b>N</b> = 567,169 <b>Population sampled:</b> General population from 6 states  <b>Excluded:</b> duplicate questionnaires, death before entry, withdrawal from the study, proxy responders, poor health, prevalent cases of cancer, end-stage renal	<b>CVD mortality</b> defined as deaths from diseases of the heart, hypertension (without heart disease), cerebrovascular diseases, atherosclerosis, aortic aneurysm, and dissection and other diseases of the arteries, arterioles, and capillaries (i.e., <b>ICD9: 390–398, 401–404, 410–438, 440–448; ICD10: I00–I09, I10–I13, I20–I51, I60–I78</b> ).	<b>g/1,000 kcal (median)</b>  <b>Females</b> <b>Q1</b> (ref): 38.5 <b>Q2:</b> 51.5 <b>Q3:</b> 61.3 <b>Q4:</b> 72.3 <b>Q5:</b> 91.1  <b>Males</b> <b>Q1</b> (ref): 33.5	<b>Females</b> <b>Q1</b> (ref): 767 <b>Q2:</b> 627 <b>Q3:</b> 641 <b>Q4:</b> 644 <b>Q5:</b> 727  <b>Males</b> <b>Q1</b> (ref): 1,631 <b>Q2:</b> 1,477 <b>Q3:</b> 1,425	<b>Model 1:</b> age and total energy intake  <b>Model 2:</b> model 1 + BMI, marital status, smoking, race, education, physical activity, and intake of vegetables, alcohol, saturated and polyunsaturated fats, history of hypertension, history of	<b>Females</b> <b>Model 1; HR (95% CI)</b> <b>Q1</b> (ref): 1 <b>Q2:</b> 0.76 (0.69, 0.85) <b>Q3:</b> 0.76 (0.68, 0.84) <b>Q4:</b> 0.75 (0.68, 0.83)  <b>Males</b> <b>Model 1; HR (95% CI)</b> <b>Q1</b> (ref): 1 <b>Q2:</b> 0.85 (0.80, 0.92) <b>Q3:</b> 0.81 (0.76, 0.87) <b>Q4:</b> 0.79 (0.74, 0.85)

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
	Public funding	disease, heart disease or stroke, diabetes, gallbladder disease, extreme energy intake (i.e., beyond twice the IQR above the 75th or below the 25th percentile of sex-specific Box-Cox transformed energy intake).  <b>n</b> = 353,751  <b>Sex:</b> females (n = 147,380), males (n = 206,371) <b>Ethnicity:</b> ~ 93% White, 3% African American, 2% Hispanic, 2% Asian/Other <b>Age:</b> 50-71 y	Deaths were ascertained by annual linkage to the US Social Security Administration Death Master File. Confirmation of the vital status and information on underlying causes of death were then obtained through follow-up searches of the National Death Index.	<u>Q2</u> : 45.7 <u>Q3</u> : 55.2 <u>Q4</u> : 65.9 <u>Q5</u> : 87.7  <b>n/person years</b> <b>Females</b> <u>Q1</u> (ref): 29,476/356,660 <u>Q2</u> : 29,477/359,619 <u>Q3</u> : 29,476/359,607 <u>Q4</u> : 29,477/359,619 <u>Q5</u> : 29,476/356,660  <b>Males</b> <u>Q1</u> (ref): 41,275/487,045 <u>Q2</u> : 41,276/495,312 <u>Q3</u> : 41,276/495,312 <u>Q4</u> : 41,276/495,312 <u>Q5</u> : 41,275/497,173  <b>Exposure assessment:</b> SFFQ	<u>Q4</u> : 1,382 <u>Q5</u> : 1,573	hypercholesterolemia, and use of aspirin	<u>Q5</u> : 0.87 (0.79, 0.97) <b>P per trend = 0.04</b>  <b>Model 2; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.91 (0.82, 1.02) <u>Q3</u> : 0.97 (0.86, 1.08) <u>Q4</u> : 0.97 (0.86, 1.09) <u>Q5</u> : 1.10 (0.96, 1.25) <b>P per trend=0.09</b>	<u>Q5</u> : 0.94 (0.88, 1.01) <b>P per trend = 0.14</b>  <b>Model 2; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.97 (0.90, 1.04) <u>Q3</u> : 0.96 (0.89, 1.04) <u>Q4</u> : 0.95 (0.88, 1.03) <u>Q5</u> : 1.08 (0.99, 1.18) <b>P per trend=0.08</b>
2	<b>Takayama<sup>a</sup></b>  Japan  Nagata et al. (2019) <sup>38</sup>  14.1 y (mean)	<b>N</b> = 34,018  <b>Population sampled:</b> General population  <b>Excluded:</b> incomplete baseline questionnaire and dietary data, prevalent cancer, stroke or CHD at baseline	<b>CVD mortality</b>  Information concerning subjects who died or moved away was obtained from residential registers or family registers. Causes of death were identified from death certificates provided by the	<b>E%, range (median)</b>  <b>Females</b> <u>Q1</u> (ref): 0.8–8.1 (6.6) <u>Q2</u> : 8.1–10.4 (9.3) <u>Q3</u> : 10.4–13.1 (11.6) <u>Q4</u> : 13.1–42.9 (15.4)  <b>Males</b> <u>Q1</u> (ref): 0.5–5.7 (4.4)	<b>Females</b> <u>Q1</u> (ref): 258 <u>Q2</u> : 215 <u>Q3</u> : 193 <u>Q4</u> : 237  <b>Males</b> <u>Q1</u> (ref): 174 <u>Q2</u> : 168	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + height, BMI, physical activity, smoking status, alcohol consumption, education, marital status and histories of diabetes and hypertension	<b>Females</b>  <b>Model 1; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.81 (0.67, 0.97) <u>Q3</u> : 0.75 (0.62, 0.90) <u>Q4</u> : 0.86 (0.72, 1.03)	<b>Males</b>  <b>Model 1; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.84 (0.68, 1.04) <u>Q3</u> : 1.03 (0.84, 1.26) <u>Q4</u> : 1.04 (0.85, 1.27)

<sup>38</sup> This study also reports on other relevant exposures, but only results on total sugars and fructose are extracted, which is in line with the approach for considering studies from the update of the literature search.

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
	Public funding	n = 29,079 Females = 15,724 Males = 13,355  Ethnicity: Asian Age: ≥35 y	Legal Affairs Bureau. CVD deaths coded as follows: <b>ICD-10: I00–I99</b>	Q2: 5.7–7.9 (6.8) Q3: 7.9–10.7 (9.1) Q4: 10.7–40.9 (13.0)  n per quartile <b>Females</b> Q1 (ref): 3,931 Q2: 3,931 Q3: 3,931 Q4: 3,931  <b>Males</b> Q1 (ref): 3,339 Q2: 3,339 Q3: 3,339 Q4: 3,338  <b>Exposure assessment:</b> SFFQ	Q3: 206 Q4: 227	<b>Model 3:</b> model 2 + total energy and intakes of fat, salt, dietary fibre and coffee	<b>P per trend =</b> 0.66  <b>Model 2; HR (95% CI)</b> Q1 (ref): 1 Q2: 0.85 (0.71, 1.02) Q3: 0.81 (0.67, 0.97) Q4: 0.89 (0.75, 1.07) <b>P per trend =</b> 0.25  <b>Model 3; HR (95% CI)</b> Q1 (ref): 1 Q2: 0.86 (0.71, 1.04) Q3: 0.84 (0.69, 1.04) Q4: 0.99 (0.81, 1.22) <b>P per trend =</b> 0.83	<b>P per trend =</b> 0.26  <b>Model 2; HR (95% CI)</b> Q1 (ref): 1 Q2: 0.87 (0.70, 1.08) Q3: 1.07 (0.87, 1.32) Q4: 1.12 (0.91, 1.38) <b>P per trend =</b> 0.08  <b>Model 3; HR (95% CI)</b> Q1 (ref): 1 Q2: 0.93 (0.74, 1.16) Q3: 1.21 (0.96, 1.52) Q4: 1.39 (1.08, 1.78) <b>P per trend =</b> 0.001
2	WHI  USA  Tasevska et al. (2018)  Up to 16 y	N = 122,970  <b>Population sampled:</b> Postmenopausal women recruited from 40 clinical centres  <b>Excluded:</b> implausible self-reported energy intake	<b>CVD incidence</b> defined as fatal and non-fatal cases of CHD, stroke, congestive heart failure, angina, coronary artery bypass graft, percutaneous coronary intervention, deep vein thrombosis, pulmonary embolism, carotid artery disease.	<b>Geometric mean (95%CI)</b>  <b>*Total sugars (g/day):</b> 93 (68, 123)  <b>Total sugars density (g/1000 kcal):</b> 61.4 (61.2, 61.5)	n = 5,802	<b>Model 1:</b> Age, energy intake (total energy intake in <b>energy substitution</b> models; non-sugars and non-alcohol energy in <b>energy partition</b> models)  <b>Model 2:</b> model 1 + race and ethnicity, education,	<b>HR (95% CI) for a 20% increase in TS<sup>42</sup></b> <b>Uncalibrated TS intake</b>  <b>Energy substitution:</b> M1: 0.96 (0.94, 0.97) M2: 0.97 (0.95, 0.99)  <b>Energy partition:</b> M1: 0.96 (0.95, 0.98) M2: 0.98 (0.96, 0.99)	

<sup>42</sup> Corresponding to 18.0 g/1,000 kcal for calibrated and 12.6 g/1,000 kcal for uncalibrated TS

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
	Public funding	(<600 or >5000kcal/day) on the FFQ, missing data on relevant covariates, prevalent cases of CVD at baseline.  <b>Follow-up rate:</b> 99.5%  <b>n</b> = 64,751  <b>Sex:</b> females <b>Ethnicity:</b> ~ 84% Caucasian, 7.6% Black, Hispanic/Latino 4% and 3% Asian/Pacific <b>Age:</b> 50-79 y	<b>Identification of incident cases:</b> by self-report in annual-biannual questionnaires.  Vital status and causes of death were ascertained by linkage with the National Death Index of the National Center of Health Statistics.  <b>Adjudication of outcome<sup>39</sup>:</b> Reports were reviewed by local physician adjudicators, who assigned diagnoses based on medical records, death certificates, and autopsy reports. These were forwarded to central physician adjudicators for independent confirmation.  <u>Positive predictive value</u> ~70% for CHD and 77% for stroke <u>Negative predictive value when events are not reported:</u> NR <u>Sensitivity:</u> NR	<b>*Calibrated<sup>40</sup> total sugars:</b> 186 (149, 245)  <b>Calibrated<sup>41</sup> total sugars density (g/1000 kcal):</b> 95.0 (94.6, 95.3)  <b>Exposure assessment:</b> SFFQ		smoking status, hormone therapy use, history of treated HTN or hypercholesterolemia, history of CVD, family history of T2DM, alcohol consumption, activity-related energy expenditure, ratio of sodium-to-potassium intake  <b>Model 3:</b> model 2 + BMI	M3: 0.98 (0.96, 1.00)	M3: 0.98 (0.97, 1.00)
							<b>Calibrated TS intake</b>	
							<b>Energy substitution:</b> M1: 0.98 (0.94, 1.03) M2: 0.97 (0.87, 1.09) M3: 0.97 (0.85, 1.12)	<b>Energy partition:</b> M1: 1.03 (0.95, 1.12) M2: 0.91 (0.80, 1.04) M3: 0.90 (0.84, 0.97)
Exposure: added sugars								
1	NIH-AARP  USA  Tasevska et al. (2014) *	Same population and exclusion criteria as for total sugars	Same ascertainment of outcome as for total sugars  <u>CVD mortality</u>	g/1,000kcal (median) Females Q1 (ref): 10.1 Q2: 15.1 Q3: 20.6 Q4: 28.6 Q5: 45.4	Females Q1(ref): 753 Q2: 652 Q3: 576 Q4: 670 Q5: 755  Males	Model 1: Age and total energy intake  Model 2: model 1+ BMI, marital status, smoking, race, education, physical activity, and intake of vegetables, alcohol,	Females  Model 1; HR (95% CI) Q1 (ref): 1 Q2: 0.85 (0.77, 0.95)	Males  Model 1; HR (95% CI) Q1 (ref): 1 Q2: 0.85 (0.79, 0.92)

<sup>39</sup> Curb JD, McTiernan A, Heckbert SR, et al. Outcomes ascertainment and adjudication methods in the Women's Health Initiative. Ann Epidemiol. 2003;13(9 suppl): S122–S128

<sup>40</sup> Calibration equations were derived for TS, energy, protein, NA/K intake ratio, and activity-related energy expenditure

<sup>41</sup> Calibration equations were derived for TS, energy, protein, NA/K intake ratio, and activity-related energy expenditure

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
	13 y  Public funding			<b>Males</b> <u>Q1</u> (ref): 9.2 <u>Q2</u> : 14.7 <u>Q3</u> : 21.0 <u>Q4</u> : 29.4 <u>Q5</u> : 47.0  <b>n/person years</b> <b>Females</b> <u>Q1</u> (ref): 29,476/356,660 <u>Q2</u> : 29,477/359,619 <u>Q3</u> : 29,476/359,607 <u>Q4</u> : 29,477/359,619 <u>Q5</u> : 29,476/356,660  <b>Males</b> <u>Q1</u> (ref): 41,275/490,815 <u>Q2</u> : 41,276/495,312 <u>Q3</u> : 41,276/495,312 <u>Q4</u> : 41,276/495,312 <u>Q5</u> : 41,275/497,173  <b>Exposure assessment:</b> SFFQ	<u>Q1</u> (ref): 1,643 <u>Q2</u> : 1,435 <u>Q3</u> : 1,406 <u>Q4</u> : 1,443 <u>Q5</u> : 1,561	saturated and polyunsaturated fats, history of hypertension, history of hypercholesterolemia, and use of aspirin	<u>Q3</u> : 0.75 (0.67, 0.84) <u>Q4</u> : 0.89 (0.80, 0.99) <u>Q5</u> : 1.10 (1.00, 1.22) <b>P per trend = 0.0003</b>  <b>Model 2; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.94 (0.84, 1.04) <u>Q3</u> : 0.82 (0.72, 0.92) <u>Q4</u> : 0.94 (0.84, 1.05) <u>Q5</u> : 0.96 (0.86, 1.08) <b>P per trend=0.94</b>	<u>Q3</u> : 0.83 (0.77, 0.89) <u>Q4</u> : 0.86 (0.80, 0.92) <u>Q5</u> : 1.01 (0.95, 1.09) <b>P per trend = 0.04</b>  <b>Model 2; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.91 (0.85, 0.98) <u>Q3</u> : 0.87 (0.82, 0.95) <u>Q4</u> : 0.87 (0.81, 0.94) <u>Q5</u> : 0.91 (0.84, 0.98) <b>P per trend=0.07</b>
2	<b>Mr and Ms OS</b>  China  Liu et al. (2018)*  11.1 y (median)	<b>N</b> = 4,000  <b>Population sampled:</b> General population  <b>Excluded:</b> unable to walk independently, bilateral hip replacement, prevalent diabetes at baseline.  <b>Follow-up rate:</b> 74.95%	<b>CVD mortality.</b>  Data on mortality statistics were obtained from the Death Registry of the Department of Health of Hong Kong. CV causes of death were identified by the cause of death reported on the death certificate and classified according to the <b>ICD-10 codes from 100 to 199.</b>	<b>E%, median (range)</b> <u>Q1</u> (ref): 0.67 (0-1.12) <u>Q2</u> : 1.59 (1.12-2.03) <u>Q3</u> : 2.50 (2.03-3.07) <u>Q4</u> : 3.88 (3.07-4.99) <u>Q5</u> : 6.86 (4.99-54.9)  <b>n/person years</b> <u>Q1</u> (ref): 683/3,682 <u>Q2</u> : 683/3,736	<u>Q1</u> (ref): 38 <u>Q2</u> : 39 <u>Q3</u> : 31 <u>Q4</u> : 36 <u>Q5</u> : 29	<b>Model 1:</b> crude  <b>Model 2:</b> age, sex, total energy intake, dietary fat, intake of fruits and vegetables, red or processed meat, Total American Heart Association risk score, education, income, smoking, coffee, green and Chinese tea, baseline body weight.	<b>Model 1; HR (95%CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.66 (0.27, 1.62) <u>Q3</u> : 0.29 (0.09, 0.89) <u>Q4</u> : 0.38 (0.13, 1.70) <u>Q5</u> : 0.19 (0.06, 0.69) <b>P per trend = 0.003</b>  <b>Model 2; HR (95%CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.75 (0.31, 1.85)	

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Public funding	n = 3416  Sex: 50.2% females Ethnicity: Asian Age: ≥65 y		Q3: 684/3,794 Q4: 683/3,813 Q5: 683/3,822  Exposure assessment: SFFQ		history of CVD, history of cancer, physical activity.  Model 3: model 2 + changes in body fat at year 4	Q3: 0.32 (0.10, 1.01) Q4: 0.45 (0.16, 1.27) Q5: 0.25 (0.07, 0.90) P per trend = 0.011  Model 3; HR (95%CI) Q1 (ref): 1 Q2: 0.69 (0.27, 1.73) Q3: 0.32 (0.10, 1.02) Q4: 0.48 (0.16, 1.47) Q5: 0.33 (0.08, 1.43) P per trend = 0.055
Exposure: free sugars							
2	Mr and Ms OS  China  Liu et al. (2018)*  11.1 y (median)  Public funding	Same population and exclusion criteria as for added sugars	Same ascertainment of outcome as for added sugars  CVD mortality	E%, median (range) Q1 (ref): 0.87 (0 – 1.61) Q2: 2.20 (1.61 – 2.80) Q3: 3.52 (2.80 – 4.31) Q4: 5.33 (4.31 – 6.55) Q5: 9.68 (6.56 – 54.9)  n/person years Q1 (ref): 682/3,666 Q2: 683/3,766 Q3: 684/3,827 Q4: 680/3,800 Q5: 680/3,822  Exposure assessment: SFFQ	Q1 (ref): 39 Q2: 32 Q3: 28 Q4: 37 Q5: 30	Model 1: crude  Model 2: age, sex, total energy intake, dietary fat, intake of fruits and vegetables, red or processed meat, Total American Heart Association risk score, education, income, smoking, coffee, green and Chinese tea, baseline body weight, history of CVD, history of cancer, physical activity.	Model 1; HR (95%CI) Q1 (ref): 1 Q2: 0.36 (0.12, 1.14) Q3: 0.53 (0.2, 1.44) Q4: 0.41 (0.14, 1.18) Q5: 0.47 (0.17, 1.28) P per trend = 0.157  Model 2; HR (95%CI) Q1 (ref): 1 Q2: 0.38 (0.12, 1.25) Q3: 0.64 (0.22, 1.88) Q4: 0.56 (0.18, 1.73) Q5: 0.69 (0.23, 2.12) P per trend = 0.577
Exposure: sucrose							
1	MDCS  Sweden	N = 28,098  Population sampled: general population from the city of Malmö	CVD incidence defined as fatal and non-fatal cases of CHD (fatal or non-fatal MI or death due to IHD; ICD-9 codes 410-414; ICD-10 I120-I125) and	E% (mean) + Q1 (ref): 4 Q2: 7 Q3: 8 Q4: 10 Q5: 14	Q1 (ref): 631 Q2: 528 Q3: 574 Q4: 545 Q5: 643	Model 1: age, sex, season, diet method version, total energy intake  Model 2: model 1 + BMI, smoking, alcohol intake,	Model 1; HR (95%CI) Q1 (ref): 1 Q2: 0.86 (0.76, 0.96) Q3: 0.95 (0.85, 1.06) Q4: 0.90 (0.80, 1.01) Q5: 1.11 (0.99, 1.24)



ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
	Sonestedt et al. (2015)  Up to 14 y  Public funding	<b>Excluded:</b> history of myocardial infarction, stroke, or diabetes  <b>n</b> = 26,445  <b>Sex:</b> 62% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 44-74 y	ischemic stroke (ICD-9 code 434).  CVD events were identified by linkage to the Swedish Hospital Discharge Registry and Cause-of-death Registry. Stroke events were also identified from the local stroke registry in Malmö.	<b>Person-years:</b> <u>Q1</u> (ref): 72,294 <u>Q2</u> : 73,978 <u>Q3</u> : 73,457 <u>Q4</u> : 73,527 <u>Q5</u> : 71,677  <b>Exposure assessment:</b> 7-d food record and SFFQ		leisure-time physical activity, education  <b>Excluding BMI as a covariate or additional adjustments for several dietary factors or systolic blood pressure and anti-hypertensive drug use did not influence the risk estimates (data not shown).</b>	<b>P per trend = 0.05</b>  <b>Model 2; HR (95%CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.92 (0.81, 1.03) <u>Q3</u> : 1.02 (0.91, 1.14) <u>Q4</u> : 0.94 (0.84, 1.06) <u>Q5</u> : 1.08 (0.96, 1.21) <b>P per trend = 0.18</b>	
<b>1</b>	<b>NIH-AARP</b>  USA  Tasevska et al. (2014)*  13 y  Public funding	<b>Same population and exclusion criteria as for total sugars</b>	<b>Same ascertainment of outcome as for total sugars</b>  <b><u>CVD mortality</u></b>	<b>g/1,000kcal (median)</b>  <b>Females</b> <u>Q1</u> (ref): 13.6 <u>Q2</u> : 18.6 <u>Q3</u> : 22.8 <u>Q4</u> : 27.9 <u>Q5</u> : 37.3  <b>Males</b> <u>Q1</u> (ref): 11.8 <u>Q2</u> : 16.8 <u>Q3</u> : 21.1 <u>Q4</u> : 26.2 <u>Q5</u> : 35.4  <b>n/person years</b> <b>Females</b> <u>Q1</u> (ref): 29,476/356,660 <u>Q2</u> : 29,477/359,619 <u>Q3</u> : 29,476/359,607 <u>Q4</u> : 29,477/359,619 <u>Q5</u> : 29,476/356,660	<b>Females</b> <u>Q1</u> (ref): 773 <u>Q2</u> : 677 <u>Q3</u> : 597 <u>Q4</u> : 625 <u>Q5</u> : 734  <b>Males</b> <u>Q1</u> (ref): 1,659 <u>Q2</u> : 1,457 <u>Q3</u> : 1,403 <u>Q4</u> : 1,422 <u>Q5</u> : 1,547	<b>Model 1:</b> Age and total energy intake  <b>Model 2:</b> model 1 + BMI, marital status, smoking, race, education, physical activity, and intake of vegetables, alcohol, saturated and polyunsaturated fats, history of hypertension, history of hypercholesterolemia, and use of aspirin  <i>Similar results as for total sucrose are reported for added sucrose (data not shown)</i>	<b>Females</b>  <b>Model 1; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.81 (0.73, 0.90) <u>Q3</u> : 0.70 (0.63, 0.78) <u>Q4</u> : 0.73 (0.66, 0.81) <u>Q5</u> : 0.89 (0.80, 0.99) <b>P per trend = 0.08</b>  <b>Model 2; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.96 (0.87, 1.07) <u>Q3</u> : 0.86 (0.77, 0.96) <u>Q4</u> : 0.88 (0.79, 0.98)	<b>Males</b>  <b>Model 1; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.83 (0.77, 0.89) <u>Q3</u> : 0.78 (0.73, 0.84) <u>Q4</u> : 0.79 (0.74, 0.85) <u>Q5</u> : 0.90 (0.84, 0.96) <b>P per trend = 0.02</b>  <b>Model 2; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.96 (0.89, 1.03) <u>Q3</u> : 0.93 (0.86, 1.00) <u>Q4</u> : 0.91 (0.85, 0.99)

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
				<b>Males</b> <u>Q1(ref):</u> 41,275/490,815 <u>Q2:</u> 41,276/495,312 <u>Q3:</u> 41,276/495,312 <u>Q4:</u> 41,276/495,312 <u>Q5:</u> 41,275/497,173  <b>Exposure assessment:</b> SFFQ			Q5: 0.95 (0.85, 1.06) <b>P per trend = 0.36</b>	Q5: 0.93 (0.86, 1.00) <b>P per trend = 0.06</b>
<b>Exposure: fructose</b>								
<b>1</b>	<b>NIH-AARP</b>  USA  Tasevska et al. (2014)*  13 y  Public funding	Same population and exclusion criteria as for total sugars	Same ascertainment of outcome as for total sugars  <u><b>CVD mortality</b></u>	<b>g/1,000kcal (median)</b>  <b>Females</b> <u>Q1 (ref):</u> 14.8 <u>Q2:</u> 20.4 <u>Q3:</u> 25.0 <u>Q4:</u> 30.3 <u>Q5:</u> 40.4  <b>Males</b> <u>Q1 (ref):</u> 12.7 <u>Q2:</u> 18.1 <u>Q3:</u> 22.5 <u>Q4:</u> 27.8 <u>Q5:</u> 37.8  <b>n/person years</b> <b>Females</b> <u>Q1(ref):</u> 29,476/356,660 <u>Q2:</u> 29,477/359,619 <u>Q3:</u> 29,476/359,607 <u>Q4:</u> 29,477/359,619 <u>Q5:</u> 29,476/356,660	<b>Females</b> <u>Q1 (ref):</u> 805 <u>Q2:</u> 636 <u>Q3:</u> 601 <u>Q4:</u> 648 <u>Q5:</u> 716  <b>Males</b> <u>Q1(ref):</u> 1,687 <u>Q2:</u> 1,487 <u>Q3:</u> 1,449 <u>Q4:</u> 1,344 <u>Q5:</u> 1,521	<b>Model 1:</b> Age and total energy intake  <b>Model 2:</b> model 1 + BMI, marital status, smoking, race, education, physical activity, and intake of vegetables, alcohol, saturated and polyunsaturated fats, history of hypertension, history of hypercholesterolemia, and use of aspirin	<b>Females</b>  <b>Model 1; HR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.74 (0.66, 0.82) <u>Q3:</u> 0.68 (0.61, 0.76) <u>Q4:</u> 0.72 (0.65, 0.80) <u>Q5:</u> 0.85 (0.76, 0.93) <b>P per trend = 0.03</b>  <b>Model 2; HR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.90 (0.81, 1.00) <u>Q3:</u> 0.89 (0.79, 0.99) <u>Q4:</u> 0.97 (0.86, 1.08)	<b>Males</b>  <b>Model 1; HR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.83 (0.78, 0.89) <u>Q3:</u> 0.80 (0.75, 0.86) <u>Q4:</u> 0.75 (0.70, 0.81) <u>Q5:</u> 0.91 (0.84, 0.97) <b>P per trend = 0.01</b>  <b>Model 2; HR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.97 (0.90, 1.04) <u>Q3:</u> 0.98 (0.91, 1.06) <u>Q4:</u> 0.94 (0.87, 1.01)

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
				<b>Males</b> <u>Q1</u> (ref): 41,275/487,045 <u>Q2</u> : 41,276/491,184 <u>Q3</u> : 41,276/495,312 <u>Q4</u> : 41,276/495,312 <u>Q5</u> : 41,275/491,173  <b>Exposure assessment:</b> SFFQ			<u>Q5</u> : 1.07 (0.95, 1.21) <b>P per trend = 0.08</b>	<u>Q5</u> : 1.08 (1.00, 1.18) <b>P per trend = 0.08</b>
2	<b>Takayama<sup>‡</sup></b>  Japan  Nagata et al. (2019) <sup>38</sup> ci-dessus  14.1 y (mean)  Public funding	Same population and exclusion criteria as for total sugar	Same ascertainment of outcome as for total sugar  <u>CVD mortality</u>	<b>E% (median)</b> <b>Females</b> <u>Q1</u> (ref): 1.2 <u>Q2</u> : 1.8 <u>Q3</u> : 2.4 <u>Q4</u> : 3.5  <b>Males</b> <u>Q1</u> (ref): 0.9 <u>Q2</u> : 1.4 <u>Q3</u> : 2.1 <u>Q4</u> : 3.4  <b>n per quartile</b> <b>Females</b> <u>Q1</u> (ref): 3,931 <u>Q2</u> : 3,931 <u>Q3</u> : 3,931 <u>Q4</u> : 3,931  <b>Males</b> <u>Q1</u> (ref): 3,339 <u>Q2</u> : 3,339 <u>Q3</u> : 3,339 <u>Q4</u> : 3,338  <b>Exposure assessment:</b> SFFQ	<b>Females</b> <u>Q1</u> (ref): 275 <u>Q2</u> : 222 <u>Q3</u> : 204 <u>Q4</u> : 202  <b>Males</b> <u>Q1</u> (ref): 219 <u>Q2</u> : 193 <u>Q3</u> : 173 <u>Q4</u> : 190	<b>Model:</b> age, height, BMI, physical activity, smoking status, alcohol consumption, education, marital status and histories of diabetes and hypertension, total energy and intakes of fat, salt, dietary fibre and coffee	<b>Females</b> <b>Model; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 0.96 (0.80, 1.16) <u>Q3</u> : 0.97 (0.80, 1.19) <u>Q4</u> : 1.03 (0.84, 1.27) <b>P per trend = 0.70</b>	<b>Males</b> <b>Model; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 1.08 (0.87, 1.33) <u>Q3</u> : 1.14 (0.92, 1.43) <u>Q4</u> : 1.31 (1.03, 1.67) <b>P per trend = 0.002</b>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
3	<b>TLGS</b>  Iran  Bahadoran et al. (2017)  6.7 y (mean)  Public funding	<b>N</b> = 15,005  <b>Population sampled:</b> general population from one district of Tehran  <b>Excluded:</b> uncomplete demographic, anthropometric, biochemical or dietary data, unusual energy intake (<800 kcal/day or >4200 kcal/day), on specific diet for HTN, diabetes or dyslipidaemia, history of CVD.  <b>Follow-up rate:</b> 99.4%  <b>n</b> = 2,369 <b>Sex:</b> 56.7% females <b>Ethnicity:</b> Caucasian <b>Age:</b> ≥19 y	<b>CVD incidence</b> defined as any CHD-related event (MI, unstable angina pectoris, angiographic-confirmed CHD), stroke (new neurological deficit that lasted at least 24 h) or CVD death (fatal MI, CHD and stroke)  <u>Non-fatal events</u> identified through annual phone calls ( <b>self-reported</b> ) plus verification through medical files and assignment by an outcome committee.  <b>PPV, NPV or sensitivity: NR</b>  <u>Death</u> was confirmed by reviewing the death certificate or medical records.	<b>%E (range)</b> <b>T1 (ref):</b> <4.5 <b>T2:</b> 4.5-7.4 <b>T3:</b> >7.4  <b>Mean (SD)</b> 6.4 (3.7)  <b>n</b> <b>T1 (ref):</b> 789 <b>T2:</b> 790 <b>T3:</b> 790  <b>Exposure assessment:</b> SFFQ	<b>T1 (ref):</b> 20 <b>T2:</b> 22 <b>T3:</b> 37	<b>Model 1:</b> crude  <b>Model 2: CVD risk score,</b> total energy intake, total fat intake  <b>CVD risk score</b> calculated according to the sex-specific algorithms that incorporate age, total cholesterol, HDL-C, SBP, treatment for HTN, smoking, diabetes status.	<b>Model 1; HR (95% CI)</b> <b>T1 (ref):</b> 1 <b>T2:</b> 1.08 (0.59, 1.98) <b>T3:</b> 1.83 (1.07, 3.16) <b>P for trend = 0.041</b>  <b>Model 2; HR (95% CI)</b> <b>T1 (ref):</b> 1 <b>T2:</b> 1.15 (0.62, 2.12) <b>T3:</b> 1.81 (1.04, 3.15) <b>P for trend = 0.068</b>  <b>HR (95% CI) per each SD increase (3.7E%)</b> <b>Model 1:</b> 1.48 (1.25, 1.75) <b>Model 2:</b> 1.35 (1.15, 1.58)
<b>Exposure: SSSD</b>							
1	<b>MDCS</b>  Sweden  Sonestedt et al. (2015)  Up to 14 y  Public funding	<b>Same population and exclusion criteria as for total sucrose</b>	<b>Same ascertainment of outcome as for total sucrose</b>  <b>CVD incidence</b>	<b>Mean (g/d) †</b> <b>Non-consumers (ref):</b> 0 <b>Qc1:</b> 26 <b>Qc2:</b> 89 <b>Qc3:</b> 306  <b>Person-years:</b> <b>Non-c (ref):</b> 164,894 <b>Qc1:</b> 67,500 <b>Qc2:</b> 67,072 <b>Qc3:</b> 65,467	<b>Non-c (ref):</b> 1,342 <b>Qc1:</b> 490 <b>Qc2:</b> 532 <b>Qc3:</b> 557	<b>Model 1:</b> age, sex, season, diet method version, energy intake  <b>Model 2:</b> model 1 + BMI, smoking, alcohol intake, leisure-time physical activity, education  <b>Excluding BMI as a covariate or additional adjustments for several dietary factors or systolic blood pressure and anti-</b>	<b>Model 1; HR (95%CI)</b> <b>Non-c (ref):</b> 1 <b>Qc1:</b> 0.89 (0.80, 0.99) <b>Qc2:</b> 1.05 (0.95, 1.16) <b>Qc3:</b> 1.04 (0.94, 1.15) <b>P per trend = 0.27</b>  <b>Model 2; HR (95%CI)</b> <b>Non-c (ref):</b> 1 <b>Qc1:</b> 0.93 (0.84, 1.03) <b>Qc2:</b> 1.06 (0.95, 1.17) <b>Qc3:</b> 1.00 (0.90, 1.10) <b>P per trend = 0.69</b>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
				Exposure assessment: 7-d food record and SFFQ		<i>hypertensive drug use did not influence the risk estimates (data not shown).</i>	
<b>Exposure: SSSD+SSFD</b>							
<b>1</b>	<b>NHS#</b>  USA  Malik et al. (2019)  Up to 34 y  Public funding	<b>N</b> = 121,700  <b>Population sampled:</b> female nurses  <b>Excluded:</b> history of CVD, diabetes mellitus or cancer, incomplete FFQ, missing SSB data, implausible intakes of total energy (<500 or >3500 kcal/d for women and <800 or >4200 kcal/d for men)  <b>n</b> = 80,647  <b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~93%+) <b>Age:</b> 30 – 55 y	<b>CVD mortality</b>  Deaths were identified from state vital statistics records and the National Death Index or by reports from next of kin or the postal authorities.  Cause of death was determined by physician review of medical records, autopsy reports, or death certificates. <b>(ICD-8 codes 390–458).</b>	<b>Servings/time Range</b> <b>C1(ref):</b> <1/mo <b>C2:</b> 1 to 4/mo <b>C3:</b> 2 to 6/wk <b>C4:</b> 1 to <2/d <b>C5:</b> ≥2/d  <b>Person-years</b> <b>C1(ref):</b> 1,127,585 <b>C2:</b> 604,268 <b>C3:</b> 522,058 <b>C4:</b> 163,412 <b>C5:</b> 84,884  <b>Serving size</b> = 355 ml  <b>Exposure assessment:</b> SFFQ	<b>C1(ref):</b> 1,883 <b>C2:</b> 972 <b>C3:</b> 829 <b>C4:</b> 293 <b>C5:</b> 162  Total: 4,139	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + smoking, alcohol intake, postmenopausal hormone use (NHS), physical activity, family history of diabetes, family history of myocardial infarction, family history of cancer, multivitamin use, ethnicity, and aspirin use  <b>Model 3:</b> model 2 + baseline history of hypertension and hypercholesterolemia; intake of whole grains, fruit, vegetables, red and processed meat; total energy; and BMI	<b>Model 1; HR (95%CI)</b> <b>C1(ref):</b> 1 <b>C2:</b> 1.07 (0.99, 1.16) <b>C3:</b> 1.19 (1.10, 1.29) <b>C4:</b> 1.46 (1.29, 1.65) <b>C5:</b> 1.84 (1.57, 2.17) <b>P per trend &lt;0.0001</b>  <b>Model 2; HR (95%CI)</b> <b>C1(ref):</b> 1 <b>C2:</b> 1.12 (1.04, 1.21) <b>C3:</b> 1.19 (1.09, 1.29) <b>C4:</b> 1.31 (1.16, 1.48) <b>C5:</b> 1.51 (1.28, 1.77) <b>P per trend &lt;0.0001</b>  <b>Model 3; HR (95%CI)</b> <b>C1(ref):</b> 1 <b>C2:</b> 1.07 (0.99, 1.16) <b>C3:</b> 1.10 (1.01, 1.20) <b>C4:</b> 1.21 (1.06, 1.37) <b>C5:</b> 1.37 (1.16, 1.62) <b>P per trend &lt;0.0001</b>  <b>A similar positive (significant) association was observed for ASB HR (95% CI)</b>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
							<p>C6 vs C1: 1.43 (1.10, 1.87)  <b>P per trend = 0.02</b>  C6 = <math>\geq 4</math> servings/d</p> <p><b>HR (95% CI) per serving/d increase</b>  <b>Model 1:</b> 1.23 (1.18, 1.28)  <b>Model 2:</b> 1.14 (1.09, 1.19)  <b>Model 3:</b> 1.11 (1.06, 1.16)  <b>A non-significant positive association was observed for ASB</b></p>
1	<b>HPFS†</b>  USA  Malik et al. (2019)  Up to 28 y  Public funding	<p><b>N</b> = 51,529</p> <p><b>Excluded:</b> history of CVD, diabetes mellitus or cancer, incomplete FFQ, missing SSB data, implausible intakes of total energy (&lt;500 or &gt;3500 kcal/d for women and &lt;800 or &gt;4200 kcal/d for men)</p> <p><b>n</b> = 37,716</p> <p><b>Sex:</b> males  <b>Ethnicity:</b> Caucasian (~90%+)  <b>Age:</b> 40 – 75 y</p>	<p><b>CVD mortality</b></p> <p>Deaths were identified from state vital statistics records and the National Death Index or by reports from next of kin or the postal authorities.</p> <p>Cause of death was determined by physician review of medical records, autopsy reports, or death certificates. <b>(ICD-9 codes 390–459)</b></p>	<p><b>Servings/time Range</b>  C1(ref): &lt;1/mo  C2: 1 to 4/mo  C3: 2 to 6/wk  C4: 1 to &lt;2/d  C5: <math>\geq 2</math>/d</p> <p><b>Person-years</b>  C1(ref): 348,582  C2: 168,005  C3: 302,337  C4: 66,398  C5: 28,035</p> <p><b>Serving size</b> = 355 ml</p> <p><b>Exposure assessment:</b> SFFQ</p>	<p>C1(ref): 1,593  C2: 736  C3: 1,122  C4: 222  C5: 84</p> <p>Total: 3,757</p>	<p><b>Model 1:</b> age</p> <p><b>Model 2:</b> model 1 + smoking, alcohol intake, physical activity, family history of diabetes, family history of myocardial infarction, family history of cancer, multivitamin use, ethnicity, and aspirin use</p> <p><b>Model 3:</b> model 2 + baseline history of hypertension and hypercholesterolemia; intake of whole grains, fruit, vegetables, red and processed meat; total energy; and BMI</p>	<p><b>HR (95% CI)</b>  <b>Model 1:</b>  C1(ref): 1  C2: 1.02 (0.94, 1.12)  C3: 1.09 (1.01, 1.17)  C4: 1.22 (1.06, 1.40)  C5: 1.33 (1.07, 1.66)  <b>P per trend = 0.0002</b></p> <p><b>Model 2:</b>  C1(ref): 1  C2: 1.06 (0.97, 1.16)  C3: 1.11 (1.03, 1.20)  C4: 1.20 (1.04, 1.38)  C5: 1.24 (1.00, 1.55)  <b>P per trend = 0.002</b></p> <p><b>Model 3:</b>  C1(ref): 1  C2: 1.04 (0.95, 1.14)  C3: 1.08 (1.00, 1.18)  C4: 1.17 (1.01, 1.35)  C5: 1.19 (0.95, 1.49)  <b>P per trend = 0.02</b></p> <p><b>A similar positive (non-significant) association was observed for ASB</b>  <b>HR (95% CI)</b></p>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
							<p>C6 vs C1: 1.21 (0.86, 1.70)  <b>P per trend = 0.23</b>  C6 = <math>\geq 4</math> servings/d</p> <p><b>HR (95% CI) per serving/d increase</b>  <b>Model 1:</b> 1.11 (1.05, 1.18)  <b>Model 2:</b> 1.08 (1.02, 1.15)  <b>Model 3:</b> 1.07 (1.01, 1.14)  <b>A non-significant positive association was observed for ASB</b></p>
2	<p><b>CTS<sup>‡</sup></b></p> <p>USA</p> <p>Pacheco et al. (2020)</p> <p>20 y</p> <p>Public funding</p>	<p><b>N</b> = 133,477</p> <p><b>Population sampled:</b> female teachers and administrators in California</p> <p><b>Excluded:</b> no consent, residents outside California, incomplete or incomprehensible questionnaires, incomplete dietary intake data, extreme caloric values (&lt;600 or &gt;5000 kcal/d), those aged <math>\geq 85</math> y at baseline, history of CVD and diabetes mellitus.</p> <p><b>n</b> = 106,178</p> <p><b>Sex:</b> females</p> <p><b>Ethnicity:</b> 87.3% Caucasian and 12.7% all other races</p> <p><b>Age (mean<math>\pm</math>SD):</b> 52.1 <math>\pm</math> 13.4 y</p>	<p><b>CVD incidence</b> defined as first occurrence of fatal or nonfatal MI, revascularization procedure (including coronary artery bypass grafting and percutaneous coronary intervention and/or percutaneous transluminal coronary angioplasty), or fatal or nonfatal stroke.</p> <p>Annual linkage with state-wide OSHPD hospitalization records, derived medical diagnoses, and in-patient procedures</p>	<p><b>Servings/time Range</b>  C1(ref): rare/never  C2: &gt;rare/never to &lt;1/wk  C3: <math>\geq 1</math> /wk to &lt;1 serving/d  C4: <math>\geq 1</math> serving/d</p> <p><b>Fl. oz/day (mean<math>\pm</math>SD)</b>  C1(ref): 0 <math>\pm</math> 0.0  C2: 2.6 <math>\pm</math> 0.0  C3: 5.5 <math>\pm</math> 0.0  C4: 13.5 <math>\pm</math> 0.1</p> <p>1 fl. oz = 29.6 ml</p> <p><b>Serving size</b> = 355 ml</p> <p><b>n per categories</b>  C1(ref): 43,425  C2: 35,422  C3: 22,825  C4: 4,506</p>	<p>C1(ref): 4,648  C2: 2,382  C3: 1,494  C4: 324</p> <p><b>Rate per 10,000 person-y</b>  C1(ref): 64.8  C2: 38.7  C3: 37.8  C4: 41.4</p>	<p><b>Model 1:</b> age</p> <p><b>Model 2:</b> model 1 + race/ethnicity, socioeconomic status, smoking status, alcohol intake, cardiovascular disease family history, physical activity, aspirin use, multivitamin use, menopausal status, menopausal hormone therapy use, oral contraceptive use, and history of hypertension.</p> <p><b>Model 3:</b> model 2 + BMI, total energy intake, and fruit and vegetable intake.</p> <p><b>Model 4:</b> age, race/ethnicity, socioeconomic status, smoking status, alcohol intake, cardiovascular disease family history, physical activity, aspirin use, menopausal status,</p>	<p><b>Model 1; HR (95%CI)</b>  C1(ref): 1  C2: 0.99 (0.95, 1.05)  C3: 1.02 (0.96, 1.08)  C4: 1.26 (1.13, 1.42)  <b>P per trend = 0.0006</b></p> <p><b>Model 2; HR (95%CI)</b>  C1(ref): 1  C2: 1.00 (0.95, 1.06)  C3: 1.01 (0.95, 1.07)  C4: 1.18 (1.05, 1.32)  <b>P per trend = 0.019</b></p> <p><b>Model 3; HR (95%CI)</b>  C1(ref): 1  C2: 1.00 (0.95, 1.05)  C3: 1.00 (0.94, 1.07)  C4: 1.16 (1.03, 1.31)  <b>P per trend = 0.052</b></p> <p><b>Model 4; HR (95%CI)</b>  C1(ref): 1  C2: 1.01 (0.96, 1.07)  C3: 1.02 (0.96, 1.09)  C4: 1.19 (1.06, 1.34)  <b>P per trend = 0.016</b></p>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
				Exposure assessment: SFFQ		menopausal hormone therapy use, history of hypertension, body mass index, and total energy intake	
3	<b>EPIC-Multicentre</b>  DK, DE, GR, FR, NL, UK, NO  Mullee et al. (2019)*  16.4 y (mean)  Public funding	<b>N</b> = 521,330  <b>Population sampled:</b> General population  <b>Excluded:</b> prevalent diabetes, cancer, heart disease or stroke at baseline, implausible dietary data, missing dietary data, incomplete follow-up  <b>Follow-up rate</b> = 98.5%  <b>n</b> = 324,980  <b>Sex:</b> 71% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 35-70 y	<b>CVD mortality</b>  Data on vital status as well as the cause and date of death were collected by EPIC centres through record linkages with cancer registries, boards of health, and death indices in Denmark, the Netherlands, Norway, and the United Kingdom or through active follow-up (inquiries by mail or telephone to municipal registries or regional health departments or to physicians or hospitals) in Germany, Greece, and France. ( <b>ICD-10 codes I00-I99</b> )	<b>Range (Servings/time)</b> C1 (ref): <1 /mo C2: 1 – 4 /mo C3: >1 – 6 /wk C4: 1 – <2 /d C5: ≥ 2 /d  <b>Serving size</b> = 250 ml  <b>Mean (SD), g/d</b> C1 (ref): 1 (1.9) C2: 20.9 (7) C3: 98 (53.8) C4: 308.4 (64.9) C5: 708.8 (283.7)  <b>n per category</b> C1 (ref): 181,131 C2: 40,376 C3: 64,178 C4: 9,371 C5: 6,746  <b>Exposure assessment:</b> SFFQ (dietary interview in GR)	C1(ref): 3,311 C2: 955 C3: 1,206 C4: 220 C5: 175	<b>Model:</b> BMI, physical activity index, educational status, alcohol consumption, smoking status and intensity, smoking duration, ever use of contraceptive pill, menopausal status, ever use of menopausal hormone therapy, intakes of total energy, red and processed meat, fruits and vegetables, coffee, fruit and vegetable juice, and stratified by age, EPIC centre, and sex.	<b>Model; HR (95%CI)</b> C1 (ref): 1 C2: 0.97 (0.90, 1.05) C3: 0.96 (0.90, 1.04) C4: 1.06 (0.92, 1.22) C5: 1.11 (0.95, 1.30) <b>P per trend = 0.16</b>  <b>A stronger positive (significant) association was observed for ASB HR (95% CI)</b> C5 vs C1: 1.52 (1.30, 1.78) <b>P per trend = &lt;.001</b>
Exposure: 100% FJ							



ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
1	MDCS  Sweden  Sonestedt et al. (2015)  Up to 14 y  Public funding	Same population and exclusion criteria as for total sucrose	Same ascertainment of outcome as for total sucrose  <u>CVD incidence</u>	Mean (g/d) † Non-consumers (ref): 0 Qc1: 11 Qc2: 87 Qc3: 235  Person-years: Non-c (ref): 157,978 Qc1: 69,283 Qc2: 69,356 Qc3: 68,316  Exposure assessment: 7-d food record and SFFQ	Non-c (ref): 1,449 Qc1: 523 Qc2: 467 Qc3: 482	Model 1: age, sex, season, diet method version, energy intake  Model 2: model 1 + BMI, smoking, alcohol intake, leisure-time physical activity, education	Model 1; HR (95%CI) Non-c (ref): 1 Qc1: 0.89 (0.81, 0.99) Qc2: 0.87 (0.79, 0.97) Qc3: 0.93 (0.84, 1.03) P per trend = 0.03  Model 2; HR (95%CI) Non-c (ref): 1 Qc1: 0.98 (0.88, 1.08) Qc2: 0.97 (0.87, 1.08) Qc3: 0.99 (0.89, 1.10) P per trend = 0.66

BMI, body mass index; CI, confidence interval; CHD, coronary heart disease; CM, clinical modification; CVD, cardiovascular disease; FFQ, food frequency questionnaire; FJ, fruit juice; h, hours; HDL-C, high density lipoprotein cholesterol; HTN, hypertension; HR, hazard ratio; HRT, hormone replacement therapy; ICD, International Classification of Diseases; IQR, interquartile range; MI, myocardial infarction; n, participants analysed; N, participants included in the cohort; NPV, negative predictive value; NR, not reported; PPV, positive predictive value; SBP, systolic blood pressure; SD, standard deviation; SFFQ, semiquantitative food frequency questionnaire; T2DM, type 2 diabetes mellitus; TS, total sugars; USA, United States of America; y, years. \*Data provided by authors. † Exposure adjusted for total energy intake using the nutrient residuals model. ‡ Study identified through an update of the literature search conducted in July 2020. Unless otherwise noted, all of the above cohorts are prospective cohorts.

## Coronary heart disease

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
Exposure: total sugars							
1	EPIC-Multicentre†	N = 521,330  Population sampled: General population	<u>CHD incidence</u>  Events identified by various methods, including primary and secondary care	g/d † Range (median) Q1 (ref): ≤77.2 (64.9) Q2: 77.3 – 93.5 (85.5) Q3: 93.6 – 108.8 (99.9)	Q1 (ref): 1,509 Q2: 1,306 Q3: 1,200 Q4: 1,181	Model 1: age, sex, and recruitment centre  Model 2: model 1 + smoking, education,	Model 1; HR (95% CI) Q1 (ref): 1 Q2: 1.05 (0.97, 1.13) Q3: 1.05 (0.97, 1.13) Q4: 1.07 (0.99, 1.16)

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	DK, DE, GR, IT, NL, UK, ES, SE  Sieri et al. (2020)*  12.8 y (median)  Public funding	<b>Excluded:</b> history of diabetes, myocardial infarction or stroke, prevalent cases of CHD, missing dietary data, top or bottom 1% of the ratio of energy intake to energy requirement, incomplete follow-up  <b>n</b> = 338,325  <b>Sex:</b> 64% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 35-70 y	databases, hospital admissions records, and self-report. Nonfatal CHD events were validated from medical records or databases. Fatalities were usually confirmed from mortality databases ( <b>ICD-9-CM 410 to 414; ICD-10-CM I20 to I25</b> )	<u>Q4</u> : 108.9 – 129.3 (116.1) <u>Q5</u> : >129.3 (144.5)  <b>n per quintile of intake</b> <u>Q1</u> (ref): 68,116 <u>Q2</u> : 68,116 <u>Q3</u> : 68,116 <u>Q4</u> : 68,116 <u>Q5</u> : 68,115  <b>Exposure assessment:</b> SFFQ	<u>Q5</u> : 1,182	physical activity, BMI, and blood pressure variable  <b>Model 3:</b> model 2 + intakes of energy, protein, alcohol, fiber, starch, saturated and monounsaturated fat	<u>Q5</u> : 1.13 (1.04, 1.23) <b>P per trend = 0.006</b>  <b>Model 2; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 1.09 (1.01, 1.18) <u>Q3</u> : 1.09 (1.01, 1.18) <u>Q4</u> : 1.12 (1.03, 1.21) <u>Q5</u> : 1.13 (1.04, 1.23) <b>P per trend = 0.007</b>  <b>Model 3; HR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 1.12 (1.03, 1.22) <u>Q3</u> : 1.14 (1.04, 1.24) <u>Q4</u> : 1.18 (1.07, 1.31) <u>Q5</u> : 1.24 (1.09, 1.40) <b>P per trend = 0.001</b>  <b>HR (95% CI) per each 50 g/d increase</b> <u>Model 1</u> : 1.05 (1.01, 1.09) <u>Model 2</u> : 1.04 (1.00, 1.08) <u>Model 3</u> : 1.09 (1.02, 1.17)

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
1	<b>SCHS</b>  Singapore  Rebello et al. (2014)  15 y (median)  Public funding	<b>N</b> = 63,257  <b>Population sampled:</b> General population of Chinese adults living in Singapore  <b>Excluded:</b> self-reported history of cancer, heart attack, angina or diabetes, extreme daily energy intake (<700 or >3,700kcal/d for men and <600 or >3000kcal/d for women, >3 SDs from the mean)  <b>n</b> = 53,469  <b>Follow-up rate:</b> 99.9%  <b>Males</b> <b>n</b> = 23,501 <b>Females</b> <b>n</b> = 29,968 <b>Ethnicity:</b> Asian <b>Age:</b> 45-74 y	<b>CHD mortality</b>  Coronary deaths identified through the population-based Singapore Registry of Births and Deaths ( <b>ICD-9 codes 410.0 to 414.9</b> ).	<b>%E, median (range)</b> <b>Females</b> <b>Q1 (ref):</b> 7.2 (0-9.2) <b>Q2:</b> 10.7 (9.2-12.1) <b>Q3:</b> 13.4 (12.1-14.8) <b>Q4:</b> 16.4 (14.8-18.4) <b>Q5:</b> 21.6 (18.4-49.1)  <b>Males</b> <b>Q1 (ref):</b> 7.3 (0-9.2) <b>Q2:</b> 10.7 (9.2-12.1) <b>Q3:</b> 13.4 (12.1-14.8) <b>Q4:</b> 16.4 (14.8-18.4) <b>Q5:</b> 21.3 (18.4-50.4)  <b>n/person-years*</b>  <b>Females</b> <b>Q1:</b> 5,469/83,065 <b>Q2:</b> 5,732/88,870 <b>Q3:</b> 5,954/92,146 <b>Q4:</b> 6,152/95,655 <b>Q5:</b> 6,661/103,183  <b>Males</b> <b>Q1:</b> 5,224/73,847 <b>Q2:</b> 4,962/72,091 <b>Q3:</b> 4,740/69,500 <b>Q4:</b> 4,542/66,784 <b>Q5:</b> 4,033/59,292  <b>Exposure assessment:</b> SFFQ	<b>Females*</b> <b>Q1 (ref):</b> 178 <b>Q2:</b> 148 <b>Q3:</b> 107 <b>Q4:</b> 104 <b>Q5:</b> 101  <b>Males*</b> <b>Q1 (ref):</b> 300 <b>Q2:</b> 208 <b>Q3:</b> 185 <b>Q4:</b> 197 <b>Q5:</b> 132	<b>Model 1:</b> age, year of interview, father's dialect and total energy intake.  <b>Model 2:</b> model 1 + smoking, alcohol consumption, sleep duration, physical activity, education level, BMI, history of hypertension, and for women only, menopausal status and hormone-replacement therapy use.  <b>Model 3:</b> model 2 + dietary cholesterol, ratio of polyunsaturated to saturated fat and fibre intake.  <i>Adjustments as specified in Model 2 did not materially change the RRs as estimated in Model 1 (not shown)</i>	<b>Females</b>  <b>Model 1; HR (95% CI)</b> <b>Q1 (ref):</b> 1.00 <b>Q2:</b> 0.94 (0.76, 1.17) <b>Q3:</b> 0.71 (0.56, 0.90) <b>Q4:</b> 0.71 (0.55, 0.90) <b>Q5:</b> 0.70 (0.55, 0.90) <b>P per trend &lt;0.01</b>  <b>Model 3; HR (95% CI)</b> <b>Q1 (ref):</b> 1.00 <b>Q2:</b> 1.03 (0.82, 1.29) <b>Q3:</b> 0.82 (0.64, 1.06) <b>Q4:</b> 0.88 (0.68, 1.14) <b>Q5:</b> 0.95 (0.72, 1.27) <b>P per trend = 0.08</b>  <b>RR (95% CI) per each 5%E</b> <b>M 1:</b> 0.86 (0.80, 0.92) <b>M 3:</b> 0.93 (0.86, 1.01)	<b>Males</b>  <b>Model 1; HR (95% CI)</b> <b>Q1 (ref):</b> 1.00 <b>Q2:</b> 0.81 (0.68, 0.97) <b>Q3:</b> 0.76 (0.63, 0.91) <b>Q4:</b> 0.83 (0.70, 1.00) <b>Q5:</b> 0.64 (0.52, 0.79) <b>P per trend &lt;0.01</b>  <b>Model 3; HR (95% CI)</b> <b>Q1 (ref):</b> 1.00 <b>Q2:</b> 0.82 (0.68, 0.98) <b>Q3:</b> 0.78 (0.64, 0.94) <b>Q4:</b> 0.84 (0.68, 1.02) <b>Q5:</b> 0.64 (0.50, 0.81) <b>P per trend &lt;0.01</b>  <b>RR (95% CI) per each 5%E</b> <b>M 1:</b> 0.90 (0.85, 0.95) <b>M 3:</b> 0.90 (0.85, 0.96)
							<b>HR (95% CI) for a 20% increase in TS<sup>45</sup></b> <b>Total CHD</b> <b>Uncalibrated TS intake</b>	
2	<b>WHI</b>  USA	<b>N</b> = 122,970  <b>Population sampled:</b>	<b>CHD incidence</b>	<b>Geometric mean (95%CI)</b>	<b>n</b> = 4,291	<b>Model 1:</b> age, energy intake (total energy intake in <b>energy</b> )		

<sup>45</sup> Corresponding to 18.0 g/1,000 kcal for calibrated and 12.6 g/1,000 kcal for uncalibrated TS

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
	Tasevska et al. (2018)  Up to 16 y  Public funding	Postmenopausal women recruited from 40 clinical centres  <b>Excluded:</b> implausible self-reported energy intake (<600 or >5000kcal/day) on the FFQ, missing data on relevant covariates, prevalent cases of CVD at baseline.  <b>Follow-up rate:</b> 99.5%  <b>n</b> = 64,751  <b>Sex:</b> females <b>Ethnicity:</b> ~ 84% Caucasian, 7.6% Black, Hispanic/Latino 4% and 3% Asian/Pacific <b>Age:</b> 50-79 y	<b>Identification of incident cases:</b> by self-report in annual-biannual questionnaires.  Vital status and causes of death were ascertained by linkage with the National Death Index of the National Center of Health Statistics.  <b>Adjudication of outcome<sup>43</sup>:</b> Reports were reviewed by local physician adjudicators, who assigned diagnoses on the basis of medical records, death certificates, and autopsy reports. These were forwarded to central physician adjudicators for independent confirmation. Cases of angina, congestive heart failure, and revascularization were centrally adjudicated to search for unreported MI.  <u>Positive predictive value</u> @ 70% for CHD <u>Negative predictive value when events are not reported:</u> NR  <u>Sensitivity:</u> NR	<b>*Total sugars (g/day):</b> 93 (68, 123)  <b>Total sugars density (g/1000 kcal):</b> 61.4 (61.2, 61.5)  <b>*Calibrated total sugars:</b> 186 (149, 245)  <b>Calibrated<sup>44</sup> total sugars density (g/1000 kcal):</b> 95.0 (94.6-95.3)  <b>Exposure assessment:</b> SFFQ		<b>substitution</b> models; non-sugars and non-alcohol energy in <b>energy partition</b> models)  <b>Model 2:</b> model 1 + race and ethnicity, education, smoking status, hormone therapy use, history of treated HTN or hypercholesterolemia, history of CVD, family history of T2DM, alcohol consumption, activity-related energy expenditure, ratio of sodium-to-potassium intake  <b>Model 3:</b> model 2 + BMI	<b>Energy substitution:</b> <u>M1:</u> 0.95 (0.93, 0.97) <u>M2:</u> 0.97 (0.95, 0.99) <u>M3:</u> 0.97 (0.95, 1.00)	<b>Energy partition:</b> <u>M1:</u> 0.96 (0.94, 0.97) <u>M2:</u> 0.98 (0.96, 0.99) <u>M3:</u> 0.98 (0.96, 1.00)
							<b>Calibrated TS intake</b>	
							<b>Energy substitution:</b> <u>M1:</u> 0.99 (0.94, 1.04) <u>M2:</u> 0.96 (0.86, 1.07) <u>M3:</u> 0.96 (0.83, 1.11)	<b>Energy partition:</b> <u>M1:</u> 1.05 (0.95, 1.15) <u>M2:</u> 0.90 (0.78, 1.04) <u>M3:</u> 0.89 (0.81, 0.96)
							<b>Non-fatal MI</b> <b>Uncalibrated TS intake</b>	
							<b>Energy substitution:</b> <u>M1:</u> 0.95 (0.92, 0.97) <u>M2:</u> 0.98 (0.94, 1.02) <u>M3:</u> 0.98 (0.95, 1.02)	<b>Energy partition:</b> <u>M1:</u> 0.95 (0.93, 0.98) <u>M2:</u> 0.99 (0.96, 1.02) <u>M3:</u> 0.99 (0.96, 1.02)
							<b>Calibrated TS intake</b>	
							<b>Energy substitution:</b> <u>M1:</u> 0.97 (0.93, 1.02) <u>M2:</u> 0.96 (0.85, 1.09) <u>M3:</u> 0.96 (0.81, 1.14)	<b>Energy partition:</b> <u>M1:</u> 1.00 (0.90, 1.11) <u>M2:</u> 0.87 (0.76, 0.98) <u>M3:</u> 0.87 (0.78, 0.97)
							<b>Fatal CHD</b> <b>Uncalibrated TS intake</b>	
							<b>Energy substitution:</b> <u>M1:</u> 0.93 (0.90, 0.97) <u>M2:</u> 0.96 (0.91, 1.02) <u>M3:</u> 0.97 (0.92, 1.03)	<b>Energy partition:</b> <u>M1:</u> 0.94 (0.91, 0.97) <u>M2:</u> 0.97 (0.93, 1.02) <u>M3:</u> 0.98 (0.94, 1.02)
							<b>Calibrated TS intake</b>	
<b>Energy substitution:</b> <u>M1:</u> 0.91 (0.87, 0.96) <u>M2:</u> 0.94 (0.73, 1.20)	<b>Energy partition:</b> <u>M1:</u> 0.91 (0.80, 1.04) <u>M2:</u> 0.97 (0.78, 1.20) <u>M3:</u> 0.93 (0.79, 1.09)							

<sup>43</sup> Curb JD, McTiernan A, Heckbert SR, et al. Outcomes ascertainment and adjudication methods in the Women's Health Initiative. Ann Epidemiol. 2003;13(9 suppl): S122–S128

<sup>44</sup> Calibration equations were derived for TS, energy, protein, NA/K intake ratio, and activity-related energy expenditure

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
							M3: 0.93 (0.70, 1.25)
<b>Exposure: sucrose</b>							
1	<b>MDCS</b>  Sweden  Warfa et al. (2016)  17 y  Public funding	<b>N</b> = 28,098  <b>Population sampled:</b> general population from the city of Malmö  <b>Excluded:</b> history of myocardial infarction, stroke, or diabetes, missing data on relevant covariates (smoking, physical activity, education)  <b>n</b> = 26,190  <b>Sex:</b> 62% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 45-73 y	<b>CHD incidence</b>  CHD events were identified by linkage to the Swedish Hospital Discharge Registry and Cause-of-death Registry.  <b>CHD</b> = ICD-9 codes 410-414 (ICD-10 I120-I125)	<b>E%, (range)</b> <u>C1</u> (ref): <5 <u>C2</u> : 5-7.5 <u>C3</u> : 7.5-10 <u>C4</u> : 10-15 <u>C5</u> : >15  <b>n/person-years:</b> <u>C1</u> (ref): 3,284/56,249 <u>C2</u> : 7,516/132,605 <u>C3</u> : 7,717/135,942 <u>C4</u> : 6,374/110,476 <u>C5</u> : 1,299/21,859  <b>Exposure assessment:</b> 7-d food record and SFFQ	<u>C1</u> (ref): 343 <u>C2</u> : 681 <u>C3</u> : 699 <u>C4</u> : 605 <u>C5</u> : 165	<b>Model 1:</b> age, sex, method of data collection, season of data collection and total energy intake  <b>Model 2:</b> model 1 + smoking status, alcohol consumption, leisure-time physical activity, educational level, and intakes of fruits and vegetables, wholegrains, coffee, fermented milk, meat and fish.  <b>Model 3:</b> model 2 + WC	<b>Model 1 ; HR (95%CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 0.91 (0.80, 1.04) <u>C3</u> : 0.93 (0.82, 1.06) <u>C4</u> : 0.98 (0.86, 1.12) <u>C5</u> : 1.48 (1.22, 1.78) <b>P per trend &lt;0.001</b>  <b>Model 2 ; HR (95%CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 0.97 (0.85, 1.11) <u>C3</u> : 1.02 (0.89, 1.16) <u>C4</u> : 1.00 (0.87, 1.15) <u>C5</u> : 1.34 (1.11, 1.63) <b>P per trend = 0.01</b>  <b>Model 3 ; HR (95%CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 0.99 (0.87, 1.13) <u>C3</u> : 1.03 (0.90, 1.18) <u>C4</u> : 1.02 (0.89, 1.18) <u>C5</u> : 1.37 (1.13, 1.66) <b>P per trend = 0.008</b>
<b>Exposure: SSSD</b>							
1	<b>MDCS</b>  Sweden  Sonestedt et al. (2015)  Up to 14 y  Public funding	<b>Same population and exclusion criteria as for total sucrose</b>	<b>Same ascertainment of outcome as for total sucrose</b>  <b>CHD incidence</b>	<b>Mean (g/d) †</b> <u>Non-consumers</u> (ref): 0 <u>Qc1</u> : 26 <u>Qc2</u> : 89 <u>Qc3</u> : 306  <b>Person-years:</b> <u>Non-c</u> (ref): 164,894 <u>Qc1</u> : 67,500 <u>Qc2</u> : 67,072 <u>Qc3</u> : 65,467	NR	<b>Model 1:</b> age, sex, season, diet method version, energy intake  <b>Model 2:</b> model 1 + BMI, smoking, alcohol intake, leisure-time physical activity, education  <b>Excluding BMI as a covariate or additional adjustments</b>	<b>Model 2; HR (95%CI)</b> <u>Non-c</u> (ref): 1 <u>Qc1</u> : 0.98 (0.85, 1.12) <u>Qc2</u> : 1.05 (0.92, 1.20) <u>Qc3</u> : 1.02 (0.89, 1.16) <b>P per trend = 0.59</b>  <b>Results for CHD only reported for model 2</b>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
				Exposure assessment: 7-d food record and SFFQ		for several dietary factors or systolic blood pressure and anti-hypertensive drug use did not influence the risk estimates (data not shown).	
<b>Exposure: SSSD+SSFD</b>							
2	HPP#  (ARIC, ATBC, HPFS, IWH, WHS, NHS80, NHS86)  USA  Keller et al. (2020)  8.2 y (median)  Public funding	N = 284,345  <b>Population sampled:</b> Nurses, health professionals and general population  <b>Excluded:</b> history of CVD, diabetes, cancer, extreme energy intake  n = 284,345  <b>Sex:</b> 76.1% females <b>Ethnicity:</b> Majority Caucasian <b>Age:</b> ≥ 35 y	<b>CHD incidence</b>  Standardized criteria, questionnaires supplemented by medical records, autopsy reports or death certificates reviewed by physicians were used to ascertain CHD events and death in each study. CHD events refers to any first incident CHD event, first event can be fatal CHD, and CHD death refers to total incident CHD death.	ml/d, median 47.9  <b>SSBs categories</b> C1(ref): <1 serving/d C2: 1-2 servings/d C3: >2 servings/d  <b>n per SSB category</b> C1(ref): 261,169 C2: 13,463 C3: 8,791  <b>Serving size</b> = 355 ml  <b>Exposure assessment:</b> SFFQ	<b>Total CHD events:</b> n=4,258  <b>Events per category of intake NR</b>	<b>Model 1:</b> smoking, physical activity, education and alcohol  <b>Model 2:</b> model 1 + fiber, trans-fat, poly-unsaturated fat/saturated fat ratio  <b>Model 3:</b> model 2 + total energy  <b>Model 4:</b> model 3 + BMI  <b>Model 5:</b> Model 4 + baseline hypertension and high cholesterol	<b>Model 1; HR (95%CI)</b> C1(ref): 1 C2: 1.20 (1.05, 1.39) C3: 1.50 (1.23, 1.82)  <b>Model 2; HR (95%CI)</b> C1(ref): 1 C2: 1.18 (1.03, 1.37) C3: 1.17 (0.97, 1.42)  <b>Model 3; HR (95%CI)</b> C1(ref): 1 C2: 1.14 (0.99, 1.32) C3: 1.12 (0.92, 1.36)  <b>Model 4; HR (95%CI)</b> C1(ref): 1 C2: 1.15 (1.00, 1.33) C3: 1.10 (0.90, 1.33)  <b>Model 5; HR (95%CI)</b> C1(ref): 1 C2: 1.12 (0.97, 1.29) C3: 1.14 (0.93, 1.40)  <b>HR (95%CI) per 355 ml/d increase</b> <b>Model 1:</b> 1.16 (1.11, 1.22) <b>Model 2:</b> 1.08 (1.03, 1.14) <b>Model 3:</b> 1.09 (1.04, 1.15) <b>Model 4:</b> 1.08 (1.02, 1.14) <b>Model 5:</b> 1.08 (1.02, 1.13)

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
2	<p><b>CTS<sup>‡</sup></b></p> <p>USA</p> <p>Pacheco et al. (2020)</p> <p>20 y</p> <p>Public funding</p>	<p><b>N</b> = 133,477</p> <p><b>Excluded:</b> no consent, residents outside California, incomplete or incomprehensible questionnaires, incomplete dietary intake data, extreme caloric values (&lt;600 or &gt;5000 kcal/d), those aged ≥85 y at baseline, history of CVD and diabetes mellitus.</p> <p><b>n</b> = 106,178</p> <p><b>Sex:</b> females</p> <p><b>Ethnicity:</b> 87.3% Caucasian and 12.7% all other races</p> <p><b>Age (mean±SD):</b> 52.1 ± 13.4 y</p>	<p><b>CHD incidence</b> defined as first occurrence of fatal or nonfatal MI</p> <p>Annual linkage with state-wide OSHPD hospitalization records, derived medical diagnoses, and in-patient procedures</p>	<p><b>Servings/time Range</b></p> <p><u>C1(ref):</u> rare/never</p> <p><u>C2:</u> &gt;rare/never to &lt;1 serving per week</p> <p><u>C3:</u> ≥1 serving/wk to &lt;1 serving/d</p> <p><u>C4:</u> ≥1 serving/d</p> <p><b>Fl. oz/day (mean±SD)</b></p> <p><u>C1(ref):</u> 0 ± 0.0</p> <p><u>C2:</u> 2.6 ± 0.0</p> <p><u>C3:</u> 5.5 ± 0.0</p> <p><u>C4:</u> 13.5 ± 0.1</p> <p>1 fl. oz = 29.6 ml</p> <p><b>Serving size</b> = 355 ml</p> <p><b>n per categories</b></p> <p><u>C1(ref):</u> 43,425</p> <p><u>C2:</u> 35,422</p> <p><u>C3:</u> 22,825</p> <p><u>C4:</u> 4,506</p> <p><b>Exposure assessment:</b> SFFQ</p>	<p><u>C1(ref):</u> 1,441</p> <p><u>C2:</u> 681</p> <p><u>C3:</u> 460</p> <p><u>C4:</u> 95</p> <p><b>Rate per 10,000 person-y</b></p> <p><u>C1(ref):</u> 19.6</p> <p><u>C2:</u> 10.9</p> <p><u>C3:</u> 11.5</p> <p><u>C4:</u> 12.0</p>	<p><b>Model 1:</b> age</p> <p><b>Model 2:</b> model 1 + race/ethnicity, socioeconomic status, smoking status, alcohol intake, cardiovascular disease family history, physical activity, aspirin use, multivitamin use, menopausal status, menopausal hormone therapy use, oral contraceptive use, and history of hypertension.</p> <p><b>Model 3:</b> model 2 + body mass index, total energy intake, and fruit and vegetable intake.</p> <p><b>Model 4:</b> age, race/ethnicity, socioeconomic status, smoking status, alcohol intake, cardiovascular disease (CVD) family history, physical activity, aspirin use, menopausal status, menopausal hormone therapy use, history of hypertension, body</p>	<p><b>Model 1; HR (95%CI)</b></p> <p><u>C1(ref):</u> 1</p> <p><u>C2:</u> 0.95 (0.87, 1.04)</p> <p><u>C3:</u> 1.06 (0.95, 1.18)</p> <p><u>C4:</u> 1.26 (1.02, 1.55)</p> <p><b>P per trend</b> = 0.022</p> <p><b>Model 2; HR (95%CI)</b></p> <p><u>C1(ref):</u> 1</p> <p><u>C2:</u> 0.96 (0.87, 1.05)</p> <p><u>C3:</u> 1.05 (0.94, 1.16)</p> <p><u>C4:</u> 1.14 (0.92, 1.40)</p> <p><b>P per trend</b> = 0.148</p> <p><b>Model 3; HR (95%CI)</b></p> <p><u>C1(ref):</u> 1</p> <p><u>C2:</u> 0.95 (0.87, 1.06)</p> <p><u>C3:</u> 1.04 (0.93, 1.16)</p> <p><u>C4:</u> 1.15 (0.92, 1.43)</p> <p><b>P per trend</b> = 0.154</p> <p><b>Model 4; HR (95%CI)</b></p> <p><u>C1(ref):</u> 1</p> <p><u>C2:</u> 0.98 (0.89, 1.07)</p> <p><u>C3:</u> 1.07 (0.96, 1.19)</p> <p><u>C4:</u> 1.18 (0.95, 1.47)</p> <p><b>P per trend</b> = 0.060</p>



ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
						mass index, and total energy intake	
3	<b>EPIC-Multicentre</b>  DK, DE, GR, FR, NL, UK, NO  Mullee et al. (2019)*  16.4 y (mean)  Public funding	<b>N</b> = 521,330  <b>Population sampled:</b> General population  <b>Excluded:</b> prevalent diabetes, cancer, heart disease or stroke at baseline, implausible dietary data, missing dietary data, incomplete follow-up  <b>Follow-up rate</b> = 98.5%  <b>n</b> = 324,980  <b>Sex:</b> 71% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 35-70 y	<b>CHD mortality</b>  Data on vital status as well as the cause and date of death were collected by EPIC centers through record linkages with cancer registries, boards of health, and death indices in Denmark, the Netherlands, Norway, and the United Kingdom or through active follow-up (inquiries by mail or telephone to municipal registries or regional health departments or to physicians or hospitals) in Germany, Greece, and France. ( <b>ICD-10 codes I20-I25</b> )	<b>Servings/time (Range)</b> <u>C1 (ref):</u> <1 /mo <u>C2:</u> 1 – 4 /mo <u>C3:</u> >1 – 6 /wk <u>C4:</u> ≥ 1 /d  <b>Serving size</b> = 250 ml  <b>g/d, mean (SD)</b> <u>C1 (ref):</u> 1 (1.9) <u>C2:</u> 20.9 (7) <u>C3:</u> 98 (53.8) <u>C4:</u> 477.9 (275)  <b>n per category</b> <u>C1 (ref):</u> 178,971 <u>C2:</u> 39,798 <u>C3:</u> 63,426 <u>C4:</u> 15,881  <b>Exposure assessment:</b> SFFQ (dietary interview in GR)	<u>C1 (ref):</u> 1,151 <u>C2:</u> 377 <u>C3:</u> 454 <u>C4:</u> 159	<b>Model:</b> BMI, physical activity index, educational status, alcohol consumption, smoking status and intensity, smoking duration, ever use of contraceptive pill, menopausal status, ever use of menopausal hormone therapy, intakes of total energy, red and processed meat, fruits and vegetables, coffee, fruit and vegetable juice, and stratified by age, EPIC centre, and sex.	<b>Model; HR (95%CI)</b> <u>C1 (ref):</u> <u>C2:</u> 1.03 (0.91, 1.16) <u>C3:</u> 0.95 (0.85, 1.07) <u>C4:</u> 1.04 (0.87, 1.23) <b>P per trend</b> = 0.84  <b>A stronger positive (significant) association was observed for ASB HR (95% CI)</b> <u>C4 vs C1:</u> 1.41 (1.11, 1.79) <b>P per trend</b> = 0.003
3	<b>REGARDS</b> <sup>†</sup>  USA  Collin et al. (2019)* <sup>46</sup>  6 y (mean)	<b>N</b> = 30,183  <b>Population sampled:</b> General population  <b>Excluded:</b> self-reported history of CHD, T2DM, stroke or transient ischemic attack at	<b>CHD mortality</b>  <b>Identification and ascertainment of cases:</b> hospital medical records; interviews with family members, reports in annual-biannual questionnaires or those calling the study toll-free number and where	<b>Range (E%)</b> <u>C1(ref):</u> 0 - <5 <u>C2:</u> 5 - <10 <u>C3:</u> >10  <b>E% Median (IQR)</b> 1.3 (0.2, 6.1)  <b>g/day</b>	<u>C1(ref):</u> 39 <u>C2:</u> 29 <u>C3:</u> 100  Total: n=168	<b>Model 1:</b> unadjusted  <b>Model 2:</b> age, race, sex, education, smoking and alcohol  <b>Model 3:</b> model 2+ BMI	<b>Model 1; HR (95%CI)</b> <u>C1:</u> ref. <u>C2:</u> 1.38 (0.91, 2.09) <u>C3:</u> 1.81 (1.25, 2.62)  <b>Model 2; HR (95%CI)</b> <u>C1:</u> ref. <u>C2:</u> 1.17 (0.76, 1.81) <u>C3:</u> 1.80 (1.21, 2.67)

<sup>46</sup> Collin et al., 2019 also reports on the exposure 100% FJ, however, these results were not extracted, which is in line with the approach applied for considering studies from the update of the literature search.



ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Public funding	baseline, missing dietary data  <b>Follow-up rate:</b> 75.3%  <b>n</b> = 13,440  <b>Sex:</b> 40.7% females  <b>Ethnicity:</b> Caucasian 68.9%, African-America 31.1%  <b>Age:</b> ≥45 y	death certificates, and the National Death Index were used to identify date and cause of death; death events identified through searches of the Social Security Administration Master Death File.  Adjudication was then done by clinicians (general internists, cardiologists, and physician assistants) who had undergone specific training to identify causes of death. This group reviewed dates and causes of death by examining death certificates, medical records, and other administrative databases.	<b>Median (IQR)</b> 50.5 (6.0, 232.2)  <b>N per category of intake = NR</b>  <b>Exposure assessment:</b> SFFQ		<b>Model 4:</b> model 3+ dietary and physical activity	<b>Model 3; HR (95%CI)</b> <u>C1</u> : ref. <u>C2</u> : 1.18 (0.77, 1.82) <u>C3</u> : 1.78 (1.19, 2.65)  <b>Model 4; HR (95%CI)</b> <u>C1</u> : ref. <u>C2</u> : 1.08 (0.70, 1.67) <u>C3</u> : 1.59 (1.06, 2.40)  <b>A non-significant, positive association was observed for total sugary beverages (combination of SSBs and 100%FJ) HR (95% CI)</b> <u>C3 vs C1</u> : 1.44 (0.97, 2.15)  <b>HR (95%CI) per 355 ml increase</b> <b>Model 4:</b> 1.11 (0.90, 1.39)  <b>A similar positive (non-significant) association was observed for sugary beverages in the continuous analysis.</b>
<b>Exposure: SSSD+SSFD+SSFJ</b>							

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
1	JPHC	N = 43,149	<u>CHD incidence</u>	Range (servings/week) C1 (ref): 0 C2: 1-2 C3: 3-4 C4: 5-7	Females C1 (ref): 53 C2: 25 C3: 11 C4: 4	Model 1: age	Females	Males
	Japan	Population sampled: General population	Identification and ascertainment of cases: hospital record review.	Serving size: 250 g	Males C1 (ref): 155 C2: 112 C3: 49 C4: 44	Model 2: model 1 + history of HTN, history of diabetes, smoking status, ethanol intake, physical activity, job status, and intakes of seafood, meat, fruit, and sodium	Model 1; OR (95%CI) C1 (ref): 1 C2: 0.89 (0.56, 1.44) C3: 1.49 (0.78, 2.86) C4: 1.14 (0.41, 3.16) P per trend = 0.34	Model 1; OR (95%CI) C1 (ref): 1 C2: 0.77 (0.61, 0.99) C3: 0.75 (0.55, 1.04) C4: 1.11 (0.79, 1.55) P per trend = 0.34
	Eshak et al. (2012)	Excluded: self-reported stroke or cancer at baseline, kidney disease or chronic liver disease; missing baseline data for the exposure, implausible total energy intake (<500 or >3500 kcal/d)	CHD: criteria of the Monitoring Trends and Determinants of Cardiovascular Disease project, which requires evidence from electrocardiograms, cardiac enzymes, or autopsy.	n/person-years Females C1 (ref): 11,820/194,873 C2: 6,401/107,883 C3: 1,769/29,376 C4: 921/14,892		Model 3: model 2 + BMI and total energy intake	Model 3; OR (95%CI) C1 (ref): 1 C2: 0.96 (0.59, 1.55) C3: 1.52 (0.78, 2.95) C4: 0.88 (0.30, 2.60) P per trend = 0.52	Model 3; OR (95%CI) C1 (ref): 1 C2: 0.85 (0.66, 1.08) C3: 0.85 (0.61, 1.18) C4: 1.04 (0.74, 1.48) P per trend = 0.37
	Up to 18 y	Follow-up rate: 98%		Males C1 (ref): 7,453/112,327 C2: 6,535/105,686 C3: 3,000/48,366 C4: 1,886/30,199		Adjustments as specified in Model 2 did not materially change the RRs as estimated in Model 3		
	Public funding	n = 39,786 Males: 18,875 Females: 20,911		Exposure assessment: SFFQ				
		Ethnicity: Asian Age: 40-59 y						
Exposure: 100% FJ								

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
1	<b>MDCS</b>  Sweden  Sonestedt et al. (2015)  Up to 14 y  Public funding	Same population and exclusion criteria as for total sucrose	CHD incidence Same ascertainment of outcome as for total sucrose	Mean (g/d) † Non-consumers (ref): 0 Qc1: 11 Qc2: 87 Qc3: 235  Person-years: Non-c (ref): 157,978 Qc1: 69,283 Qc2: 69,356 Qc3: 68,316  Exposure assessment: 7-d food record and SFFQ	NR	<b>Model 1:</b> age, sex, season, diet method version, energy intake  <b>Model 2:</b> model 1 + BMI, smoking, alcohol intake, leisure-time physical activity, education  <i>Excluding BMI as a covariate or additional adjustments for several dietary factors or systolic blood pressure and anti-hypertensive drug use did not influence the risk estimates (data not shown).</i>	<b>Model 2; HR (95%CI)</b> Non-c (ref): 1 Qc1: 0.97 (0.85, 1.11) Qc2: 0.91 (0.79, 1.05) Qc3: 1.02 (0.89, 1.17) <b>P per trend = 0.77</b>  <i>Results for CHD only reported for model 2</i>

BMI, body mass index; BMR, basal metabolic rate; CHD, coronary heart disease; CI, confidence interval; CVD, cardiovascular disease; CM, clinical modification; HDL, high density lipoprotein; HR, hazard ratio; HRT, hormone replacement therapy; HTN, hypertension; ICD, International Classification of Disease; MI, myocardial infarction; n, participants analysed; N, participants included in the cohort; NR, not reported; RR, risk ratio; SBP, systolic blood pressure; SD, standard deviation; SFFQ, semiquantitative food frequency questionnaire; T2DM, type 2 diabetes mellitus; TS, total sugars; USA, United States of America; WC, waist circumference; y, years. \* Data provided by authors. † Exposure adjusted for total energy intake using the nutrient residuals model. ‡ Study identified through an update of the literature search conducted in July 2020. Unless otherwise noted, all of the above cohorts are prospective cohorts.

## Stroke

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
Exposure: total sugars							

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
1	EPIC-Morgen  The Netherlands  Burger et al. (2011)  11.9 y (mean)  Public funding	N = 22,654  <b>Population sampled:</b> General population  <b>Excluded:</b> subjects with no consent to linkage with disease registries, history of T2D or CVD, missing nutritional data and/or ranked in the top or bottom 0.5% of the ratio of reported energy intake over estimated BMR  n = 19,608  <b>Males</b> n = 8,855  <b>Females</b> n = 10,753 <b>Ethnicity:</b> Caucasian <b>Age:</b> 20-65 y	<b>Stroke incidence</b> (ischemic and haemorrhagic)  Data on morbidity obtained from register of discharge diagnosis from all hospitals ( <b>ICD-9-CM 430 to 434, 436</b> )  Information on vital status obtained through linkage with municipal administration registries. Causes of death obtained from Statistics Netherlands ( <b>ICD-9-CM 430 to 434, 436; ICD-10-CM I60 to I66</b> )	<b>g/day, mean (SD)</b> <b>Females:</b> 111.7 (29.6) <b>Males:</b> 105.7 (29.1)  <b>Exposure assessment:</b> SFFQ	<b>Females</b> 109  <b>Males</b> 120	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + smoking, education, BMI, physical activity, HRT and OC use  <b>Model 3:</b> model 2 + total energy intake and energy-adjusted alcohol, vitamin C, dietary fibre and saturated and monounsaturated fat, starch  <b>Model 4:</b> model 3 + plasma total cholesterol and HDL-cholesterol	<b>Females</b>  <b>HR (95% CI) per each SD increase (29.5g/d)</b> <b>Model 1:</b> 0.93 (0.77, 1.12) <b>Model 2:</b> 0.96 (0.80, 1.16) <b>Model 3:</b> 0.96 (0.65, 1.44) <b>Model 4:</b> 0.95 (0.63, 1.42)  <b>Males</b>  <b>HR (95% CI) per each SD increase (29.5g/d)</b> <b>Model 1:</b> 0.96 (0.80, 1.15) <b>Model 2:</b> 0.99 (0.83, 1.19) <b>Model 3:</b> 1.00 (0.70, 1.44) <b>Model 4:</b> 1.01 (0.70, 1.46)	
1	EPICOR  Italy  Sieri et al. (2013)  10.9 y (mean)  Public funding	N = 47,749  <b>Population sampled:</b> General population  <b>Excluded:</b> prevalent CVD at recruitment, subjects unavailable for follow-up at time 0, incomplete dietary of lifestyle questionnaires, ratio of total energy intake BMR at either extreme of the distribution (first and last half percentiles, prevalent diabetes, missing	<b>Stroke incidence</b> (ischemic and haemorrhagic) Suspected events were identified from mortality files ( <b>ICD-10 codes I60–I69; ICD-10 codes E10–E14, I10–I15, I46, I49, and I70</b> ) and assigned after verification against hospital discharge and clinical records. Suspected cases identified from hospital discharge databases ( <b>ICD9-CM codes 342, 430–434, or 436–438, or by procedure codes for carotid</b>	<b>g/d (median)</b> <b>Q1 (ref):</b> 69 <b>Q2:</b> 90 <b>Q3:</b> 104 <b>Q4:</b> 120 <b>Q5:</b> 150  <b>n</b> <b>Q1 (ref):</b> 8,826 <b>Q2:</b> 8,813 <b>Q3:</b> 8,819 <b>Q4:</b> 8,808 <b>Q5:</b> 8,833  <b>Exposure assessment:</b> SFFQ	<b>Total stroke</b> <b>Q1 (ref):</b> 77 <b>Q2:</b> 64 <b>Q3:</b> 70 <b>Q4:</b> 59 <b>Q5:</b> 85  <b>Ischemic stroke</b> <b>Q1 (ref):</b> 43 <b>Q2:</b> 41 <b>Q3:</b> 36 <b>Q4:</b> 32 <b>Q5:</b> 43	<b>Model 1:</b> sex, type of FFQ, age  <b>Model 2:</b> model 1 + education, smoking, BMI, alcohol, non-alcohol energy intake, cereal fiber intake, saturated fat, monounsaturated fat, polyunsaturated fat and physical activity	<b>Total stroke</b> <b>Model 1; RR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 1.00 (0.68, 1.47) <b>Q3:</b> 0.91 (0.61, 1.35) <b>Q4:</b> 0.83 (0.56, 1.25) <b>Q5:</b> 1.31 (0.90, 1.90) <b>P per trend = 0.161</b>  <b>Model 2; RR (95% CI)</b> <b>Q1 (ref):</b> 1 <b>Q2:</b> 1.16 (0.78, 1.73) <b>Q3:</b> 1.09 (0.72, 1.64) <b>Q4:</b> 0.99 (0.65, 1.53) <b>Q5:</b> 1.42 (0.93, 2.16) <b>P per trend = 0.156</b>	

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
		values for confounding variables.  <b>Follow-up rate:</b> 99.6%  <b>n</b> = 44,099  <b>Sex:</b> 69% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 35-75 y	revascularization) and verified against MRI or CT scans		<b>Haemorrhagic stroke</b> <u>Q1</u> (ref): 14 <u>Q2</u> : 13 <u>Q3</u> : 18 <u>Q4</u> : 14 <u>Q5</u> : 24		<b>RR (95% CI) per each SD increase (34.4g/d)*</b> <u>Model 1</u> : 1.07 (0.95, 1.21) <u>Model 2</u> : 1.06 (0.93, 1.21)	
							<b>Ischemic stroke</b> <b>Model 1; RR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 1.02 (0.62, 1.69) <u>Q3</u> : 0.75 (0.43, 1.30) <u>Q4</u> : 0.84 (0.49, 1.42) <u>Q5</u> : 1.11 (0.67, 1.84) <b>P per trend = 0.789</b>  <b>Model 2; RR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 1.22 (0.73, 2.04) <u>Q3</u> : 0.90 (0.51, 1.58) <u>Q4</u> : 0.96 (0.55, 1.70) <u>Q5</u> : 1.09 (0.61, 1.94) <b>P per trend = 0.958</b>  <b>RR (95% CI) per each SD increase (34.4g/d)*</b> <u>Model 1</u> : 1.01 (0.85, 1.19) <u>Model 2</u> : 0.97 (0.81, 1.17)	<b>Haemorrhagic stroke</b> <b>Model 1; RR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 1.13 (0.48, 2.64) <u>Q3</u> : 1.22 (0.53, 2.80) <u>Q4</u> : 0.78 (0.31, 1.96) <u>Q5</u> : 1.74 (0.79, 3.80) <b>P per trend = 0.174</b>  <b>Model 2; RR (95% CI)</b> <u>Q1</u> (ref): 1 <u>Q2</u> : 1.18 (0.50, 2.81) <u>Q3</u> : 1.35 (0.57, 3.19) <u>Q4</u> : 0.87 (0.33, 2.27) <u>Q5</u> : 1.83 (0.77, 4.39) <b>P per trend = 0.195</b>  <b>RR (95% CI) per each SD increase (34.4g/d)*</b> <u>Model 1</u> : 1.21 (0.97, 1.51) <u>Model 2</u> : 1.23 (0.95, 1.59)

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
1	<b>EPIC-Utrecht</b>  The Netherlands  Beulens et al. (2007)*  9 y (mean)  Public funding	<b>N</b> = 17,357  <b>Population sampled:</b> Breast cancer screening participants  <b>Excluded:</b> not consent to linkage with vital status registries, missing questionnaires, energy intake of <500 kcal/day or >6,000 kcal/day, prevalent CHD, cerebrovascular disease, or diabetes.  <b>n</b> = 15,714  <b>Sex:</b> females <b>Ethnicity:</b> Caucasian <b>Age:</b> 49-70 y	<b>Stroke incidence</b> (ischemic and haemorrhagic) (ICD-9-CM 430 to 438).  Morbidity data: from the Dutch Centre for Health Care Information (standardized computerized register of hospital discharge diagnoses). Information on vital status: linkage with the municipal administration registries. Causes of death: from the women's general practitioners and coded by 2 independent physicians.	<b>g/day, mean (SD)</b> <b>Q1</b> (ref): 75 (22) <b>Q2:</b> 100 (22) <b>Q3:</b> 116 (26) <b>Q4:</b> 140 (37)  <b>n/person years</b> <b>Q1</b> (ref): 3,928/35,278 <b>Q2:</b> 3,929/35,429 <b>Q3:</b> 3,929/35,504 <b>Q4:</b> 3,928/35,423  <b>Exposure assessment:</b> SFFQ	<b>Q1</b> (ref): 63 <b>Q2:</b> 61 <b>Q3:</b> 58 <b>Q4:</b> 61	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + hypertension, cholesterolemia, smoking, BMI, SBP, physical activity, menopausal status, HRT use, OC use, alcohol intake, total energy intake, energy-adjusted intake of vitamin E; protein, dietary fiber, folate; saturated fat; and poly- and monounsaturated fat	<b>Model 1; HR (95% CI)</b> <b>Q1</b> (ref): 1 <b>Q2:</b> 0.87 (0.61, 1.23) <b>Q3:</b> 0.78 (0.55, 1.12) <b>Q4:</b> 0.79 (0.55, 1.13)  <b>Model 2; HR (95% CI)</b> <b>Q1</b> (ref): 1 <b>Q2:</b> 1.03 (0.69, 1.54) <b>Q3:</b> 0.95 (0.59, 1.55) <b>Q4:</b> 1.00 (0.52, 1.92)
2	<b>WHI</b>  USA  Tasevska et al. (2018)  Up to 16 y  Public funding	<b>N</b> = 122,970  <b>Population sampled:</b> Postmenopausal women recruited from 40 clinical centres  <b>Excluded:</b> implausible self-reported energy intake (<600 or >5000 kcal/day) on the FFQ, missing data on relevant covariates, prevalent cases of CVD at baseline.  <b>Follow-up rate:</b> 99.5%	<b>Stroke incidence</b> (ischemic and haemorrhagic) <b>Identification of incident cases:</b> by self-report in annual-biannual questionnaires.  Vital status and causes of death were ascertained by linkage with the National Death Index of the National Center of Health Statistics.  <b>Adjudication of outcome</b> <sup>47</sup> : Reports were reviewed by local physician adjudicators,	<b>Geometric mean (95%CI)</b>  <b>*Total sugars (g/day):</b> 93 (68, 123)  <b>Total sugars density (g/1000 kcal):</b> 61.4 (61.2, 61.5)  <b>*Calibrated total sugars:</b> 186 (149, 245)	<b>Total stroke</b>  <b>n</b> = 1,868  <b>Ischemic stroke</b>  <b>n</b> = 1,418  <b>Haemorrhagic stroke</b>  <b>n</b> = 314	<b>Model 1:</b> Age, energy intake (total energy intake in <b>energy substitution</b> models; non-sugars and non-alcohol energy in <b>energy partition</b> models)  <b>Model 2:</b> model 1 + race and ethnicity, education, smoking status, hormone therapy use, history of treated HTN or hypercholesterolemia, history of CVD, family history of T2DM, alcohol consumption, activity-	<b>HR (95% CI) for a 20% increase in TS</b> <b>Total stroke</b> <b>Uncalibrated TS intake</b>  <b>Energy substitution:</b> <b>M1:</b> 0.98 (0.95, 1.01) <b>M2:</b> 0.99 (0.95, 1.03) <b>M3:</b> 1.00 (0.96, 1.03)  <b>Energy partition:</b> <b>M1:</b> 0.98 (0.96, 1.00) <b>M2:</b> 0.99 (0.96, 1.02) <b>M3:</b> 0.99 (0.96, 1.02)  <b>Calibrated TS intake</b>  <b>Energy substitution:</b> <b>M1:</b> 0.96 (0.92, 1.01) <b>M2:</b> 1.00 (0.85, 1.18) <b>M3:</b> 1.00 (0.84, 1.20)  <b>Energy partition:</b> <b>M1:</b> 0.98 (0.91, 1.05) <b>M2:</b> 0.97 (0.85, 1.10)

<sup>47</sup> Curb JD, McTiernan A, Heckbert SR, et al. Outcomes ascertainment and adjudication methods in the Women's Health Initiative. Ann Epidemiol. 2003;13(9 suppl): S122-S128

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		<p>n = 64,751</p> <p><b>Sex:</b> females</p> <p><b>Ethnicity:</b> ~ 84% Caucasian, 7.6% Black, Hispanic/Latino 4% and 3% Asian/Pacific</p> <p><b>Age:</b> 50-79 y</p>	<p>who assigned diagnoses based on medical records, death certificates, and autopsy reports. These were forwarded to central physician adjudicators for independent confirmation.</p> <p><u>PPV @ 77% for stroke</u> <u>NPV when events are not reported:</u> NR</p> <p><u>Sensitivity:</u> NR</p>	<p><b>Calibrated<sup>48</sup> total sugars density (g/1000 kcal):</b> 95.0 (94.6-95.3)</p> <p><b>Exposure assessment:</b> SFFQ</p>		<p>related energy expenditure, ratio of sodium-to-potassium intake</p> <p><b>Model 3:</b> model 2 + BMI</p> <p><i>Findings for ischemic and haemorrhagic stroke are reported in Web Table 1 (not shown).</i></p> <p><i>Similar results as for total stroke are reported for ischemic and haemorrhagic stroke (data not shown).</i></p>	<p>M3: 0.95 (0.86, 1.06)</p>
<b>Exposure: sucrose</b>							
<b>1</b>	<p><b>MDCS</b></p> <p>Sweden</p> <p>Sonestedt et al. (2015)</p> <p>Up to 14 y</p> <p>Public funding</p>	<p><b>N</b> = 28,098</p> <p><b>Population sampled:</b> general population from the city of Malmö</p> <p><b>Excluded:</b> history of myocardial infarction, stroke, or diabetes</p> <p><b>n</b> = 26,445</p> <p><b>Sex:</b> 62% females</p> <p><b>Ethnicity:</b> Caucasian</p> <p><b>Age:</b> 45-73 y</p>	<p><b><u>Ischemic stroke incidence</u></b></p> <p>Events were identified by linkage to the Swedish Hospital Discharge Registry and Cause-of-death Registry and from the local stroke registry in Malmö. <b>Ischemic stroke</b> was defined as ICD-9 code 434 and confirmed based on computed tomography or autopsy.</p>	<p><b>E% (mean) †</b> Q1 (ref): 4 Q2: 7 Q3: 8 Q4: 10 Q5: 14</p> <p><b>Person-years:</b> Q1 (ref): 72,294 Q2: 73,978 Q3: 73,457 Q4: 73,527 Q5: 71,677</p> <p><b>Exposure assessment:</b> 7-d food record and SFFQ</p>	NR	<p><b>Model 1:</b> age, sex, season, diet method version, total energy intake</p> <p><b>Model 2:</b> model 1+ BMI, smoking, alcohol intake, leisure-time physical activity, education</p> <p><b>Results for stroke only reported for model 2 in the publication</b></p> <p><b>Excluding BMI as a covariate or additional adjustments</b></p>	<p><b>Model 2; HR (95%CI)</b> Q1 (ref): 1 Q2: 0.92 (0.77, 1.11) Q3: 0.93 (0.77, 1.12) Q4: 0.94 (0.78, 1.14) Q5: 0.94 (0.77, 1.14) <b>P per trend = 0.66</b></p>

<sup>48</sup> Calibration equations were derived for TS, energy, protein, NA/K intake ratio, and activity-related energy expenditure

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
						<i>for several dietary factors or systolic blood pressure and anti-hypertensive drug use did not influence the risk estimates (data not shown).</i>	
<b>Exposure: SSSD</b>							
<b>1</b>	<b>HPFS</b>  USA  Bernstein et al. (2012)  Up to 22 y  Mixed funding	<b>N</b> = 51,529  <b>Population sampled:</b> male health professionals  <b>Excluded:</b> excessive items blank on the baseline FFQ, implausibly low or high energy intakes, previously diagnosed cancer, diabetes, angina, myocardial infarction, stroke, or other CVD, including a history of PCI <sup>49</sup> or CABG <sup>50</sup> .  <b>n</b> = 43,371  <b>Sex:</b> males <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 40-75 y	<b>Stroke incidence</b> (ischemic and haemorrhagic) <b>Ischemic stroke; haemorrhagic stroke</b>  Non-fatal cases: self-reported plus confirmation through medical records (80%). 20% were probable cases (no medical records available).  <b>PPV, NPV or sensitivity: NR</b>  Deaths were identified from state vital records or the National Death Index or were reported by next of kin or the postal system. Follow-up for > 98% complete. Stroke was confirmed as fatal only if medical records were available (68%). 32% were probable (no medical records).	<b>Servings/time (range)</b> <u>Non-consumers (ref):</u> 0 <u>Qc1:</u> 0-1/wk <u>Qc2:</u> 1/wk-1/d <u>Qc3:</u> ≥1/d  <b>Serving size</b> = 12oz (355mL)  <b>Person-years</b> <u>Non-consumers (ref):</u> 259,630 <u>Qc1:</u> 204,418 <u>Qc2:</u> 323,569 <u>Qc3:</u> 54,153  <b>Exposure assessment:</b> SFFQ	<b>Total stroke</b> <u>Non-c (ref):</u> 464 <u>Qc1:</u> 381 <u>Qc2:</u> 499 <u>Qc3:</u> 72  <b>Ischemic stroke</b> <u>Non-c (ref):</u> 288 <u>Qc1:</u> 231 <u>Qc2:</u> 281 <u>Qc3:</u> 43  <b>Haemorrhagic stroke</b> <u>Non-c (ref):</u> 71 <u>Qc1:</u> 46 <u>Qc2:</u> 92 <u>Qc3:</u> 8	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + calendar time, intakes of red meat, poultry, fish, nuts, whole- and low-fat dairy products, and fruit and vegetables, alcohol intake, <i>trans</i> -fat intake, smoking, parental history of early myocardial infarction, multivitamin use, aspirin use at least once per week, vitamin E supplement use, physical exercise, ASB  <b>Model 3:</b> model 2 + BMI and energy intake  <b>Model 4:</b> model 3 + HTN  <b>Model 5:</b> model 3 + diabetes  <b>Model 6:</b> model 3 + HTN and diabetes	<b>Total stroke</b> <b>Model 1; RR (95% CI)</b> <u>Non-consumers (ref):</u> 1 <u>Qc1:</u> 0.94 (0.82, 1.09) <u>Qc2:</u> 1.02 (0.89, 1.16) <u>Qc3:</u> 1.18 (0.92, 1.53) <b>P per trend = 0.11</b>  <b>Model 2; RR (95% CI)</b> <u>Non-consumers (ref):</u> 1 <u>Qc1:</u> 0.93 (0.80, 1.07) <u>Qc2:</u> 0.99 (0.86, 1.13) <u>Qc3:</u> 1.07 (0.82, 1.40) <b>P per trend = 0.43</b>  <b>Ischemic stroke</b> <b>Model 3; RR (95% CI)</b> <u>Non-consumers (ref):</u> 1 <u>Qc1:</u> 0.90 (0.75, 1.08) <u>Qc2:</u> 0.89 (0.74, 1.06) <u>Qc3:</u> 1.02 (0.72, 1.45) <b>P per trend = 0.98</b>  <b>A positive (non-significant) association was observed for ASB HR (95% CI)</b> <u>Qc3 vs non-consumers:</u> 1.10 (0.87, 1.38) <b>P per trend = 0.24</b>  <b>Haemorrhagic stroke</b>

<sup>49</sup> Percutaneous coronary intervention

<sup>50</sup> coronary artery bypass grafting



ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
						<p><b>Adjustments as specified in models 4 and 5 did not materially change the RRs as estimated in Models 3 or 6 (not shown)</b></p> <p><i>Results for models 4 to 6 are not reported for the exposure as continuous variable</i></p> <p><i>Only results for model 3 are reported for ischemic and haemorrhagic stroke</i></p>	<p><b>Model 3; RR (95% CI)</b>  <u>Non-consumers (ref):</u> 1  <u>Qc1:</u> 0.93 (0.80, 1.08)  <u>Qc2:</u> 0.99 (0.86, 1.14)  <u>Qc3:</u> 1.08 (0.82, 1.41)  <b>P per trend = 0.43</b></p> <p><b>Model 6; RR (95% CI)</b>  <u>Non-consumers (ref):</u> 1  <u>Qc1:</u> 0.93 (0.80, 1.08)  <u>Qc2:</u> 0.99 (0.86, 1.14)  <u>Qc3:</u> 1.05 (0.80, 1.38)  <b>P per trend = 0.52</b></p> <p><b>A similar positive (non-significant) association was observed for ASB HR (95% CI)</b>  <u>Qc3 vs non-consumers:</u> 1.03 (0.86, 1.23)  <b>P per trend = 0.42</b></p> <p><b>RR (95% CI) for 1 serving/d</b></p> <p><b>Total stroke</b>  <u>Model 1:</u> 1.16 (0.97, 1.40)  <u>Model 2:</u> 1.08 (0.89, 1.31)</p>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
							<p>Model 3: 1.08 (0.89, 1.32)  <b>A similar positive association was observed for ASB</b></p> <p><b>Ischemic stroke</b>  Model 3: 1.00 (0.77, 1.30)  <b>A non-significant positive association was observed for ASB</b></p> <p><b>Haemorrhagic stroke</b>  Model 3: 1.10 (0.66, 1.81)  <b>A similar non-significant positive association was observed for ASB</b></p>	
1	<b>MDCS</b>  Sweden  Sonestedt et al. (2015)  Up to 14 y  Public funding	Same population and exclusion criteria as for total sucrose	<b>Ischemic stroke incidence</b> Same ascertainment of outcome as for total sucrose	<b>g/d (mean)</b> Non-consumers (ref): 0 Qc1: 26 Qc2: 89 Qc3: 306  <b>Person-years:</b> Non-c (ref): 164894 Qc1: 67,500 Qc2: 67,072 Qc3: 65,467  <b>Exposure assessment:</b> 7-d food record and SFFQ	NR	<b>Model 1:</b> age, sex, season, diet method version, energy intake  <b>Model 2:</b> model 1 + BMI, smoking, alcohol intake, leisure-time physical activity, education  <b>Results for stroke only reported for model 2</b>  <b>Excluding BMI as a covariate or additional adjustments for several dietary factors or systolic blood pressure and anti-hypertensive drug use did not influence the risk estimates (data not shown).</b>	<b>Model 2; HR (95%CI)</b> Non-c (ref): 1 Qc1: 0.87 (0.74, 1.02) Qc2: 1.06 (0.91, 1.24) Qc3: 0.97 (0.81, 1.13) <b>P per trend = 1.00</b>	
1	<b>NHS</b>	N = 121,700	<b>Stroke incidence</b> (ischemic and haemorrhagic)	<b>Servings/time (range)</b>	<b>Total stroke</b>	<b>Model 1:</b> age	<b>Total stroke</b>	<b>Ischemic stroke</b>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
	USA  Bernstein et al. (2012)  Up to 28 y  Mixed funding	<p><b>Population sampled:</b> female nurses</p> <p><b>Excluded:</b> excessive items blank on the baseline FFQ, implausibly low or high energy intakes, previously diagnosed cancer, diabetes, angina, myocardial infarction, stroke, or other CVD, including a history of PCI<sup>51</sup> or CABG<sup>52</sup>.</p> <p><b>n</b> = 84,085</p> <p><b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~93%+) <b>Age:</b> 30-55 y</p>	<p><b>Non-fatal cases:</b> self-reported through biannual questionnaires plus confirmation through medical records (69%). 31% were probable cases (no medical records available).</p> <p><b>PPV, NPV or sensitivity: NR</b></p> <p><b>Deaths</b> were identified from state vital records or the National Death Index or were reported by next of kin or the postal system. Follow-up for &gt; 98% complete. Stroke was confirmed as fatal only if medical records were available (58%). 42% were probable (no medical records)</p>	<p><u>Non-c (ref):</u> none <u>Qc1:</u> 0-1/wk <u>Qc2:</u> 1/wk-1/d <u>Qc3:</u> ≥1/d</p> <p><b>Serving size</b> = 12oz (355mL)</p> <p><b>Person-Years</b> <u>Non-c (ref):</u> 717,209 <u>Qc1:</u> 632,223 <u>Qc2:</u> 693,974 <u>Qc3:</u> 144,825</p> <p><b>Exposure assessment:</b> SFFQ</p>	<p><u>Non-c (ref):</u> 918 <u>Qc1:</u> 950 <u>Qc2:</u> 896 <u>Qc3:</u> 174</p> <p><b>Ischemic stroke</b> <u>Non-c (ref):</u> 462 <u>Qc1:</u> 508 <u>Qc2:</u> 463 <u>Qc3:</u> 80</p> <p><b>Haemorrhagic stroke</b> <u>Non-c (ref):</u> 181 <u>Qc1:</u> 152 <u>Qc2:</u> 156 <u>Qc3:</u> 30</p>	<p><b>Model 2:</b> model 1 + calendar time, intakes of red meat, poultry, fish, nuts, whole- and low-fat dairy products, and fruit and vegetables, alcohol intake, <i>trans</i>-fat intake, smoking, parental history of early myocardial infarction, multivitamin use aspirin use at least once per week, vitamin E supplement use, menopausal status, physical exercise, ASB</p> <p><b>Model 3:</b> model 2 + BMI and energy intake</p> <p><b>Model 4:</b> model 3 + HTN</p> <p><b>Model 5:</b> model 3 + diabetes</p> <p><b>Model 6:</b> model 3 + HTN and diabetes</p> <p><i>Adjustments as specified in models 4 and 5 did not materially change the RRs as estimated in Models 6 and 3, respectively (not shown)</i></p>	<p><b>Model 1; RR (95% CI)</b> <u>Non-c (ref):</u> 1 <u>Qc1:</u> 0.99 (0.90, 1.09) <u>Qc2:</u> 1.17 (1.07, 1.19) <u>Qc3:</u> 1.47 (1.25, 1.74) <b>P per trend &lt;0.0001</b></p> <p><b>Model 2; RR (95% CI)</b> <u>Non-c (ref):</u> 1 <u>Qc1:</u> 0.91 (0.80, 1.11) <u>Qc2:</u> 1.12 (1.02, 1.24) <u>Qc3:</u> 1.25 (1.05, 1.48) <b>P per trend = 0.004</b></p> <p><b>Model 3; RR (95% CI)</b> <u>Non-c (ref):</u> 1 <u>Qc1:</u> 1.00 (0.91, 1.10) <u>Qc2:</u> 1.11 (1.00, 1.22) <u>Qc3:</u> 1.19 (1.00, 1.42) <b>P per trend = 0.02</b></p>	<p><b>Model 3; RR (95% CI)</b> <u>Non-c (ref):</u> 1 <u>Qc1:</u> 1.05 (0.92, 1.20) <u>Qc2:</u> 1.18 (1.02, 1.35) <u>Qc3:</u> 1.28 (0.99, 1.65) <b>P per trend = 0.04</b></p> <p><b>A non-significant positive association was observed for ASB HR (95% CI)</b> <u>Qc3 vs non-consumers:</u> 1.15 (0.97, 1.35) <b>P per trend = 0.17</b></p> <p><b>Haemorrhagic stroke</b></p> <p><b>Model 3; RR (95% CI)</b> <u>Non-c (ref):</u> 1 <u>Qc1:</u> 0.95 (0.75, 1.19) <u>Qc2:</u> 1.00 (0.79, 1.26) <u>Qc3:</u> 0.85 (0.56, 1.29) <b>P per trend = 0.54</b></p>

<sup>51</sup> Percutaneous coronary intervention

<sup>52</sup> coronary artery bypass grafting

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
							<p><b>Model 6; RR (95% CI)</b>  <u>Non-c (ref): 1</u>  <u>Qc1: 1.00 (0.90, 1.10)</u>  <u>Qc2: 1.09 (0.98, 1.20)</u>  <u>Qc3: 1.14 (0.96, 1.36)</u>  <b>P per trend = 0.08</b></p> <p><b>A similar positive (non-significant) association was observed for ASB</b>  <b>HR (95% CI)</b>  <u>Qc3 vs non-consumers: 1.11 (0.98, 1.24)</u>  <b>P per trend = 0.07</b></p> <p><b>RR (95% CI) for 1 serving/d</b></p> <p><b>Total stroke</b>  <u>Model 1: 1.34 (1.21, 1.49)</u>  <u>Model 2: 1.17 (1.05, 1.30)</u>  <u>Model 3: 1.14 (1.02, 1.27)</u>  <b>A similar positive association was observed for ASB</b></p> <p><b>Ischemic stroke</b>  <u>Model 3: 1.19 (1.01, 1.39)</u>  <b>A non-significant positive association was observed for ASB</b></p> <p><b>Haemorrhagic stroke</b>  <u>Model 3: 0.92 (0.71, 1.20)</u>  <b>ASB was associated with a greater risk of haemorrhagic stroke</b></p>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
Exposure: SSSD+SSFD								
1	<b>Framingham Offspring</b>  USA  Pase et al. (2017)  Up to 10 y  Public funding	<b>N</b> = 5,124  <b>Population sampled:</b> Offspring of the original cohort of the Framingham Heart Study  <b>Excluded:</b> prevalent stroke or other significant neurological disease at baseline, <45 y  <b>n</b> = 2,888  <b>Sex:</b> 54.92% females <b>Ethnicity:</b> Caucasian <b>Age:</b> ≥45 y	<b>Stroke incidence</b> (ischemic and haemorrhagic); <b>Ischemic stroke.</b>  Defined stroke as the rapid onset of focal neurological symptoms of presumed vascular origin, lasting >24 hours or resulting in death.  Identification and ascertainment of cases: hospital admissions, medical results, questionnaires during annual health status updates.  Diagnosis of stroke was determined by a review committee comprised of at least 3 Framingham Heart Study investigators, including at least two vascular neurologists. Definite diagnosis was established after reviewing all available medical records, imaging studies, and neurological reports.	<b>Range (servings/week)</b> <b>C1 (ref):</b> 0 <b>C2:</b> >0-3 <b>C3:</b> >3  <b>n per category of intake = NR</b>  <b>Serving size</b> = 355 ml  <b>Exposure assessment:</b> SFFQ  <i>Cumulative intake defined as the averaged responses across examination cycles 5, 6 and 7 over a maximum of 7 years. Data was averaged from examination cycle 7 with data from at least one other examination (5 or 6), however, average across all 3 cycles where possible (72% completed all 3).</i>  <i>Recent intake is considered baseline intake, i.e. intake at examination 7.</i>	<b>Cases per category of intake = NR</b>  <b>Recent intake</b>  <b>Total stroke:</b> Model 1: 97/2,888 Model 2: 76/2,225 Model 3: 93/2,729  <b>Ischemic stroke:</b> Model 1: 82/2,888 Model 2: 64/2,225 Model 3: 78/2,729  <b>Cumulative intake</b>  <b>Total stroke:</b> Model 1: 87/2,690 Model 2: 70/2,137 Model 3: 85/2,598	<b>Model 1:</b> age, sex, and total caloric intake  <b>Model 2:</b> Model 1 + the dietary guidelines adherence index, self-reported physical activity, and smoking status.  <b>Model 3:</b> Model 1 + SBP, treatment of hypertension, prevalent CVD, atrial fibrillation, left ventricular hypertrophy, total cholesterol, HDL-c, prevalent diabetes mellitus, and waist to hip ratio.	<b>Recent intake</b>  <b>Total stroke Model 1:</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.21 (0.78, 1.86) <b>C3:</b> 0.89 (0.44, 1.79)  <b>Model 2:</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.15 (0.71, 1.88) <b>C3:</b> 0.69 (0.29, 1.62)  <b>Model 3:</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.22 (0.78, 1.92) <b>C3:</b> 0.88 (0.43, 1.78)  <i>A positive (non-significant) association was observed for total sugary beverages (combining SSSDs, 100%FJ and FD)</i>  <b>A significant positive association was observed for ASB HR (95% CI)</b> <b>C3 vs C1:</b> 1.97 (1.10, 3.55) <i>C3 = &gt;1 serving/day</i>  <b>Ischemic stroke Model 1:</b>	<b>Cumulative intake</b>  <b>Total stroke Model 1:</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.12 (0.70, 1.79) <b>C3:</b> 0.82 (0.40, 1.69)  <b>Model 2:</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.17 (0.70, 1.97) <b>C3:</b> 0.61 (0.25, 1.49)  <b>Model 3:</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.14 (0.70, 1.85) <b>C3:</b> 0.80 (0.38, 1.67)  <i>A similar association was observed for total sugary beverages (combining SSSDs, 100%FJ and FD)</i>  <b>A non-significant positive association was observed for ASB HR (95% CI)</b> <b>C3 vs C1:</b> 1.79 (0.91, 3.52) <i>C3 = &gt;1 serving/day</i>  <b>Ischemic stroke Model 1:</b>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
					<b>Ischemic stroke:</b> Model 1: 72/2,690 Model 2: 58/2,137 Model 3: 70/2,598		<div> C1 (ref): 1  C2: 1.24 (0.77, 1.97)  C3: 0.85 (0.39, 1.86) </div> <div> <b>Model 2:</b>  C1 (ref): 1  C2: 1.11 (0.65, 1.89)  C3: 0.69 (0.27, 1.73) </div> <div> <b>Model 3:</b>  C1 (ref): 1  C2: 1.25 (0.76, 2.04)  C3: 0.84 (0.38, 1.86) </div> <div> <i>A similar association was observed for total sugary beverages (combining SSSDs, 100%FJ and FD)</i> </div> <div> <b>A significant positive association was observed for ASB HR (95% CI)</b>  C3 vs C1: 2.34 (1.24, 4.45)  C3 = &gt;1 serving/day </div>
<b>2</b>	<b>CTS#</b>  USA  Pacheco et al. (2020)  20 y	<b>N</b> = 133,477  <b>Excluded:</b> no consent, residents outside California, incomplete or incomprehensible questionnaires, incomplete dietary intake data, extreme caloric values (<600 or	<b>Stroke incidence</b> (ischemic and haemorrhagic) defined as first occurrence of fatal or nonfatal stroke.  Annual linkage with state-wide OSHPD hospitalization records, derived medical diagnoses, and in- patient procedures	<b>Servings/time (range)</b> C1(ref): rare/never C2: >rare/never to <1 serving per week C3: ≥1 serving/wk to <1 serving/d C4: ≥1 serving/d	C1(ref): 2,787 C2: 1,415 C3: 867 C4: 189  <b>Rate per 10,000 person-y</b>	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + race/ethnicity, socioeconomic status, smoking status, alcohol intake, cardiovascular disease family history, physical activity,	<b>Model 1; HR (95%CI)</b> C1(ref): 1 C2: 1.01 (0.94, 1.08) C3: 1.01 (0.93, 1.09) C4: 1.26 (1.09, 1.46) <b>P per trend = 0.017</b>  <b>Model 2; HR (95%CI)</b> C1(ref): 1

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Public funding	<p>&gt;5000 kcal/d), those aged ≥85 y at baseline, history of CVD and diabetes mellitus.</p> <p><b>n</b> = 106,178</p> <p><b>Sex:</b> females</p> <p><b>Ethnicity:</b> 87.3% Caucasian and 12.7% all other races</p> <p><b>Age (mean±SD):</b> 52.1 ± 13.4 y</p>		<p><b>Fl. oz/day (mean±SD)</b>  <b>C1(ref):</b> 0 ± 0.0  <b>C2:</b> 2.6 ± 0.0  <b>C3:</b> 5.5 ± 0.0  <b>C4:</b> 13.5 ± 0.1</p> <p>1 fl. oz = 29.6 ml</p> <p><b>Serving size</b> = 355 ml</p> <p><b>n per categories</b>  <b>C1(ref):</b> 43,425  <b>C2:</b> 35,422  <b>C3:</b> 22,825  <b>C4:</b> 4,506</p> <p><b>Exposure assessment:</b> SFFQ</p>	<p><b>C1(ref):</b> 38.2  <b>C2:</b> 22.7  <b>C3:</b> 21.7  <b>C4:</b> 23.9</p>	<p>aspirin use, multivitamin use, menopausal status, menopausal hormone therapy use, oral contraceptive use, and history of hypertension.</p> <p><b>Model 3:</b> model 2 + body mass index, total energy intake, and fruit and vegetable intake.</p> <p><b>Model 4:</b> age, race/ethnicity, socioeconomic status, smoking status, alcohol intake, cardiovascular disease (CVD) family history, physical activity, aspirin use, menopausal status, menopausal hormone therapy use, history of hypertension, body mass index, and total energy intake</p>	<p><b>C2:</b> 1.02 (0.95, 1.08)  <b>C3:</b> 1.00 (0.93, 1.08)  <b>C4:</b> 1.19 (1.03, 1.39)  <b>P per trend</b> = <b>0.076</b></p> <p><b>Model 3; HR (95%CI)</b>  <b>C1(ref):</b> 1  <b>C2:</b> 1.00 (0.94, 1.07)  <b>C3:</b> 0.99 (0.92, 1.08)  <b>C4:</b> 1.18 (1.01, 1.37)  <b>P per trend</b> = <b>0.146</b></p> <p><b>Model 4; HR (95%CI)</b>  <b>C1(ref):</b> 1  <b>C2:</b> 1.01 (0.95, 1.08)  <b>C3:</b> 1.01 (0.93, 1.09)  <b>C4:</b> 1.21 (1.04, 1.41)  <b>P per trend</b> = <b>0.056</b></p>
3	<p><b>EPIC-Multicentre#</b></p> <p>DK, DE, GR, FR, NL, UK, NO</p> <p>Mullee et al. (2019)*</p>	<p><b>N</b> = 521,330</p> <p><b>Population sampled:</b> General population</p> <p><b>Excluded:</b> prevalent diabetes, cancer, heart disease or stroke at baseline, implausible dietary data, missing dietary data, incomplete follow-up</p>	<p><b>Stroke mortality (ischemic and haemorrhagic)</b></p> <p>Data on vital status as well as the cause and date of death were collected by EPIC centers through record linkages with cancer registries, boards of health, and death indices in Denmark,</p>	<p><b>Range (Servings/time)</b>  <b>C1 (ref):</b> &lt;1 /mo  <b>C2:</b> 1 – 4 /mo  <b>C3:</b> &gt;1 – 6 /wk  <b>C4:</b> ≥ 1 /d</p> <p><b>Serving size</b> = 250 ml</p> <p><b>Mean (SD), g/d</b>  <b>C1 (ref):</b> 1 (1.9)</p>	<p><b>C1(ref):</b> 922  <b>C2:</b> 263  <b>C3:</b> 327  <b>C4:</b> 109</p>	<p><b>Model:</b> BMI, physical activity index, educational status, alcohol consumption, smoking status and intensity, smoking duration, ever use of contraceptive pill, menopausal status, ever use of menopausal hormone therapy, intakes of total energy, red and processed meat, fruits and</p>	<p><b>Model; HR (95%CI)</b>  <b>C1 (ref):</b> 1  <b>C2:</b> 0.97 (0.84, 1.12)  <b>C3:</b> 0.99 (0.87, 1.14)  <b>C4:</b> 1.19 (0.97, 1.47)  <b>P per trend</b> = <b>0.10</b></p> <p><b>A similar positive (non-significant) association was observed for ASB HR (95% CI)</b>  <b>C4 vs C1:</b> 1.24 (0.91, 1.70)  <b>P per trend</b> = <b>0.12</b></p>

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
	16.4 y (mean)  Public funding	<b>Follow-up rate</b> = 98.5%  <b>n</b> = 324,980  <b>Sex:</b> 71% females <b>Ethnicity:</b> Caucasian <b>Age:</b> 35-70 y	the Netherlands, Norway, and the United Kingdom or through active follow-up (inquiries by mail or telephone to municipal registries or regional health departments or to physicians or hospitals) in Germany, Greece, and France. <b>(ICD-10 Codes I60-I69)</b>	<u>C2:</u> 20.9 (7) <u>C3:</u> 98 (53.8) <u>C4:</u> 477.9 (275)  <b>N per category</b> <u>C1 (ref):</u> 178,742 <u>C2:</u> 39,684 <u>C3:</u> 63,299 <u>C4:</u> 15,831  <b>Exposure assessment:</b> SFFQ (dietary interview in GR)		vegetables, coffee, fruit and vegetable juice, and stratified by age, EPIC center, and sex.		
Exposure: SSSD+SSFD+SSFJ								
1	JPHC  Japan  Eshak et al. (2012)  Up to 18 y  Public funding	<b>N</b> = 43,149  <b>Population sampled:</b> General population  <b>Excluded:</b> self-reported stroke or cancer at baseline, kidney disease or chronic liver disease; missing baseline data for the exposure, implausible total energy intake (<500 or >3500kcal/d)  <b>Follow-up rate:</b> 98%  <b>n</b> = 39,786 Males: 18,875 Females: 20,911  <b>Ethnicity:</b> Asian <b>Age:</b> 40-59 y	<b>Stroke incidence</b> (ischemic and haemorrhagic) defined as first occurrence of fatal or nonfatal stroke. Identification and ascertainment of cases: hospital record review.  <b>Stroke:</b> presence of focal neurological deficits of sudden or rapid onset lasting ≥24 h, or until death. For each subtype of stroke, a definite diagnosis was established on the basis of an examination of data collected from CT scans, MRI, or autopsy.	<b>Range (servings/week)</b> <u>C1 (ref):</u> 0 <u>C2:</u> 1-2 <u>C3:</u> 3-4 <u>C4:</u> 5-7  <b>Serving size</b> = 250 g  <b>n/person-years</b> <b>Men</b> <u>C1 (ref):</u> 7,453/112,327 <u>C2:</u> 6,535/105,686 <u>C3:</u> 3,000/48,366 <u>C4:</u> 1,886/30,199  <b>Women</b> <u>C1 (ref):</u> 11,820/194,873 <u>C2:</u> 6,401/107,883 <u>C3:</u> 1,769/29,376 <u>C4:</u> 921/14,892	<b>Total stroke</b> <b>Females</b> <u>C1 (ref):</u> 431 <u>C2:</u> 242 <u>C3:</u> 74 <u>C4:</u> 42  <b>Males</b> <u>C1 (ref):</u> 513 <u>C2:</u> 385 <u>C3:</u> 151 <u>C4:</u> 84  <b>Ischemic stroke</b> <b>Females</b> <u>C1 (ref):</u> 205 <u>C2:</u> 110 <u>C3:</u> 34	<b>Model 1:</b> age  <b>Model 2:</b> model 1 + history of HT, history of diabetes, smoking status, ethanol intake, physical activity, job status, and intakes of seafood, meat, fruit, and sodium  <b>Model 3:</b> model 2 + BMI and total energy intake  <b>Adjustments as specified in Model 2 did not materially change the RRs as estimated in Model 3</b>	<b>Females</b> <b>Total Stroke</b> <b>Model 1; OR (95%CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 1.04 (0.89, 1.22) <u>C3:</u> 1.19 (0.93, 1.53) <u>C4:</u> 1.39 (1.01, 1.91) <b>P per trend = 0.003</b>  <b>Model 3; OR (95%CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 1.07 (0.91, 1.25) <u>C3:</u> 1.12 (0.87, 1.44) <u>C4:</u> 1.21 (0.88, 1.68) <b>P per trend = 0.02</b>  <b>Ischemic stroke</b> <b>Model 1; OR (95%CI)</b> <u>C1 (ref):</u> 1	<b>Males</b> <b>Total Stroke</b> <b>Model 1; OR (95%CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 0.86 (0.75, 0.98) <u>C3:</u> 0.82 (0.69, 0.99) <u>C4:</u> 0.74 (0.59, 0.96) <b>P per trend = 0.01</b>  <b>Model 3; OR (95%CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 0.89 (0.78, 1.05) <u>C3:</u> 0.90 (0.76, 1.06) <u>C4:</u> 0.76 (0.62, 1.06) <b>P per trend = 0.07</b>  <b>Ischemic stroke</b> <b>Model 1; OR (95%CI)</b> <u>C1 (ref):</u> 1 <u>C2:</u> 0.77 (0.65, 0.91)



ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
				Exposure assessment: SFFQ	<p>C4: 28</p> <p><b>Males</b> C1 (ref): 321 C2: 222 C3: 75 C4: 52</p> <p><b>Haemorrhagic stroke</b> <b>Females</b> C1 (ref): 222 C2: 130 C3: 40 C4: 13</p> <p><b>Males</b> C1 (ref): 187 C2: 162 C3: 74 C4: 31</p>	<p>C2: 1.01 (0.80, 1.28) C3: 1.19 (0.83, 1.72) C4: 2.07 (1.39, 3.08) P per trend &lt; 0.0001</p> <p><b>Model 3; OR (95%CI)</b> C1 (ref): 1 C2: 1.03 (0.82, 1.30) C3: 1.12 (0.78, 1.63) C4: 1.83 (1.22, 2.75) P per trend = 0.001</p> <p><b>Haemorrhagic stroke</b> <b>Model 1; OR (95%CI)</b> C1 (ref): 1 C2: 1.07 (0.86, 1.32) C3: 1.22 (0.87, 1.70) C4: 0.79 (0.45, 1.39) P per trend = 0.70</p> <p><b>Model 3; OR (95%CI)</b> C1 (ref): 1 C2: 1.09 (0.87, 1.36) C3: 1.13 (0.80, 1.58) C4: 0.70 (0.40, 1.20) P per trend = 0.94</p>	<p>C3: 0.60 (0.46, 0.77) C4: 0.71 (0.53, 0.95) P per trend = 0.01</p> <p><b>Model 3; OR (95%CI)</b> C1 (ref): 1 C2: 0.85 (0.71, 1.01) C3: 0.68 (0.51, 0.89) C4: 0.75 (0.53, 1.03) P per trend = 0.07</p> <p><b>Haemorrhagic stroke</b> <b>Model 1; OR (95%CI)</b> C1 (ref): 1 C2: 0.92 (0.74, 1.13) C3: 0.92 (0.70, 1.20) C4: 0.72 (0.52, 1.00) P per trend = 0.07</p> <p><b>Model 3; OR (95%CI)</b> C1 (ref): 1 C2: 1.02 (0.82, 1.26) C3: 1.03 (0.78, 1.35) C4: 0.77 (0.55, 1.08) P per trend = 0.30</p>
Exposure: 100% FJ							
1	MDCS Sweden	Same population and exclusion criteria as for total sucrose	Ischemic stroke incidence Same ascertainment of outcome as for total sucrose	Mean (g/d)† Non-consumers (ref): 0 Qc1: 11 QC2: 87	NR	Model 1: age, sex, season, diet method version, energy intake	Model 2; HR (95%CI) Non-c (ref): 1 Qc1: 0.99 (0.85, 1.16) QC2: 1.04 (0.89, 1.23) QC3: 0.95 (0.80, 1.12)

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Sonestedt et al. (2015)  Up to 14 y  Public funding			<u>Qc3</u> : 235  <b>Person-years:</b> <u>Non-c (ref)</u> : 157,978 <u>Qc1</u> : 69,283 <u>Qc2</u> : 69,356 <u>Qc3</u> : 68,316  <b>Exposure assessment:</b> 7-d food record and SFFQ		<b>Model 2:</b> model 1 + BMI, smoking, alcohol intake, leisure-time physical activity, education  <b>Results for stroke only reported for model 2</b>  <b>Excluding BMI as a covariate or additional adjustments for several dietary factors or systolic blood pressure and anti-hypertensive drug use did not influence the risk estimates (data not shown).</b>	<b>P per trend = 0.73</b>
<b>1</b>	<b>NHS</b>  USA  Joshi et al. (1999)  14 y  Public funding	<b>N</b> = 121,700  <b>Population sampled:</b> female nurses  <b>Excluded:</b> missing $\geq 10$ items on the FFQ, implausible scores for total food intake, previous diagnosis of cancer, diabetes or CVDs  <b>n</b> = 75,596 <b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~93%+) <b>Age:</b> 30 – 55 y	<b>Ischemic stroke incidence</b>  Identification and ascertainment of cases: Self-reported by participants (or next of kin if deceased), which physicians cross checked with medical records. Deaths were also obtained from postal authorities or from the National Death Index. Only strokes confirmed by medical records were included in this analysis.  Using the medical records, including imaging results, strokes were classified into ischemic (embolic or thrombotic), haemorrhagic	<b>Median servings/d of citrus fruit juices</b> <u>Q1(ref)</u> : 0 <u>Q2</u> : NR <u>Q3</u> : NR <u>Q4</u> : NR <u>Q5</u> : 1  Overall: 0.43  <b>Serving size</b> = 6 oz (177 ml)  <b>n per quintile of intake NR</b>  <b>Exposure assessment:</b> SFFQ	<b>n cases per quintile of intake NR</b>  <b>overall:</b> 366 ischemic stroke cases	<b>Model:</b> age, smoking status, alcohol, family history of myocardial infarction, BMI, vitamin supplement use, vitamin E use, physical activity, aspirin use, 7 time periods, hypertension, hypercholesterolemia, total energy intake, and postmenopausal hormone use	<b>RR (95%CI)</b> <u>Q1(ref)</u> : 1 <u>Q2</u> : 0.80 (0.58, 1.11) <u>Q3</u> : 0.77 (0.56, 1.05) <u>Q4</u> : 0.91 (0.66, 1.25) <u>Q5</u> : 0.61 (0.45, 0.84)  <b>RR (95% CI) for each one serving/d increase</b> 0.73 (0.56, 0.95)

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
1	HPFS USA Joshipura et al. (1999) 8 y Public funding	N = 51,529  <b>Population sampled:</b> male health professionals  <b>Excluded:</b> missing ≥10 items on the FFQ, implausible scores for total food intake, previous diagnosis of cancer, diabetes or CVDs  n = 38,683 <b>Sex:</b> males <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 40-75 y	<b>Ischemic stroke incidence</b>  Identification and ascertainment of cases: Self-reported by participants (or next of kin if deceased), which physicians cross checked with medical records. Deaths were also obtained from postal authorities or from the National Death Index. Only strokes confirmed by medical records were included in this analysis.  Using the medical records, including imaging results, strokes were classified into ischemic (embolic or thrombotic), haemorrhagic (subarachnoid or intracerebral) or unknown type.	<b>Median servings/d of citrus fruit juices</b> Q1(ref): 0 Q2: NR Q3: NR Q4: NR Q5: 1  Overall: 0.43  <b>Serving size</b> = 6 oz (177 ml)  n per quintile of intake NR  <b>Exposure assessment:</b> SFFQ	<b>n cases per quintile of intake NR</b>  <b>overall:</b> 204 ischemic stroke cases	<b>Model:</b> age, smoking status, alcohol, family history of myocardial infarction, BMI, vitamin supplement use, vitamin E use, physical activity, aspirin use, 4 time periods, hypertension, hypercholesterolemia and total energy intake	<b>RR (95%CI)</b> Q1(ref): 1 Q2: 0.91 (0.60, 1.39) Q3: 0.84 (0.54, 1.31) Q4: 0.85 (0.53, 1.37) Q5: 0.74 (0.49, 1.13)  <b>RR (95% CI) for each one serving/d increase</b> 0.80 (0.57, 1.13)

ASB, artificially sweetened beverages; BMI, body mass index; CABG, coronary artery bypass grafting; CHD, coronary heart disease; CI, confidence interval; CM, clinical manifestation; CT, computed tomography; CVD, cardiovascular disease; FFQ, food frequency questionnaire; HDL, high density lipoprotein; HR, hazard ratio; HRT, hormone replacement therapy; HTN, hypertension; ICD, International Classification of Diseases; MRI, magnetic resonance imaging; n, participants analysed; N, participants included in the cohort; NPV, negative predictive value; NR, not reported; PPV, positive predictive value; RR, risk ratio; SD, standard deviation; SFFQ, semiquantitative food frequency questionnaire; SSSD, sugar-sweetened soft drinks; T2DM, type 2 diabetes mellitus; TS, total sugars; USA, United States of America; y, year. \*Data provided by the authors. † Exposure adjusted for total energy intake using the nutrient residuals model. ‡ Study identified through an update of the literature search conducted in July 2020. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Other cardiovascular endpoints

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
Exposure: total sugars								
2	WHI	N = 122,970	<u>Incidence of heart failure, coronary artery bypass graft, percutaneous coronary intervention</u>	Geometric mean (95%CI)	<u>Heart failure</u>	<b>Model 1:</b> Age, energy intake (total energy intake in <b>energy substitution</b> models; non-sugars and non-alcohol energy in <b>energy partition</b> models)  <b>Model 2:</b> model 1 + race and ethnicity, education, smoking status, hormone therapy use, history of treated HTN or hypercholesterolemia, history of CVD, family history of T2DM, alcohol consumption, activity-related energy expenditure, ratio of sodium-to-potassium intake  <b>Model 3:</b> model 2 + BMI	<b>HR (95% CI) for a 20% increase in TS<sup>55</sup></b> <b><u>Heart failure</u></b> <b>Uncalibrated TS intake</b>	
	USA	<b>Population sampled:</b> Postmenopausal women recruited from 40 clinical centres	<b>Identification of incident cases:</b> by self-report in annual-biannual questionnaires. Vital status and causes of death were ascertained by linkage with the National Death Index of the National Center of Health Statistics.	<b>*Total sugars (g/day):</b> 93 (68, 123)	n = 969		<b>Energy substitution:</b> <u>M1</u> : 0.93 (0.90, 0.95) <u>M2</u> : 0.94 (0.90, 0.98) <u>M3</u> : 0.95 (0.91, 0.99)	<b>Energy partition:</b> <u>M1</u> : 0.94 (0.92, 0.97) <u>M2</u> : 0.95 (0.92, 0.99) <u>M3</u> : 0.96 (0.93, 1.00)
	Tasevska et al. (2018)	<b>Excluded:</b> implausible self-reported energy intake (<600 or >5000kcal/day) on the FFQ, missing data on relevant covariates, prevalent cases of CVD at baseline.		<b>Total sugars density (g/1000 kcal):</b> 61.4 (61.2, 61.5)	<b>CABG</b>  n = 821			
	Up to 16 y	<b>Follow-up rate:</b> 99.5%	<b>Adjudication of outcome<sup>53</sup>:</b> Cardiovascular Central Adjudication Committee responsible for review of congestive heart failure and coronary revascularization	<b>*Calibrated total sugars:</b> 186 (149, 245)	<b>PCI</b>  n = 1855		<b>Calibrated TS intake</b>	
	Public funding	n = 64,751		<b>Calibrated<sup>54</sup> total sugars density (g/1000 kcal):</b> 95.0 (94.6-95.3)			<b>Energy substitution:</b> <u>M1</u> : 0.95 (0.90, 0.99) <u>M2</u> : 0.91 (0.72, 1.14) <u>M3</u> : 0.91 (0.61, 1.37)	<b>Energy partition:</b> <u>M1</u> : 1.05 (0.92, 1.21) <u>M2</u> : 0.97 (0.71, 1.32) <u>M3</u> : 0.87 (0.72, 1.06)
		<b>Sex:</b> females <b>Ethnicity:</b> ~ 84% Caucasian, 7.6% Black, Hispanic/Latino 4% and 3% Asian/Pacific <b>Age:</b> 50-79 y		<b>Exposure assessment:</b> SFFQ			<b>CABG</b> <b>Uncalibrated TS intake</b>	
							<b>Energy substitution:</b> <u>M1</u> : 0.94 (0.91, 0.98) <u>M2</u> : 0.94 (0.90, 0.98)	<b>Energy partition:</b> <u>M1</u> : 0.95 (0.92, 0.98) <u>M2</u> : 0.95 (0.91, 0.99) <u>M3</u> : 0.95 (0.91, 0.99)

<sup>53</sup> Curb JD, McTiernan A, Heckbert SR, et al. Outcomes ascertainment and adjudication methods in the Women's Health Initiative. Ann Epidemiol. 2003;13(9 suppl): S122–S128

<sup>54</sup> Calibration equations were derived for TS, energy, protein, NA/K intake ratio, and activity-related energy expenditure

<sup>55</sup> Corresponding to 18.0 g/1,000 kcal for calibrated and 12.6 g/1,000 kcal for uncalibrated TS

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results	
							M3: 0.94 (0.90, 0.99)	
							Calibrated TS intake	
							Energy substitution: M1: 1.02 (0.97, 1.07) M2: 0.93 (0.76, 1.14) M3: 0.93 (0.67, 1.30)	Energy partition: M1: 1.14 (1.02, 1.27) M2: 0.84 (0.69, 1.03) M3: 0.83 (0.70, 0.98)
							PCI Uncalibrated TS intake	
							Energy substitution: M1: 0.95 (0.92, 0.97) M2: 0.96 (0.93, 1.00) M3: 0.97 (0.93, 1.00)	Energy partition: M1: 0.95 (0.93, 0.97) M2: 0.97 (0.94, 1.00) M3: 0.97 (0.95, 1.00)
							Calibrated TS intake	
							Energy substitution: M1: 1.02 (0.97, 1.07) M2: 0.97 (0.83, 1.12) M3: 0.97 (0.72, 1.29)	Energy partition: M1: 1.07 (0.97, 1.18) M2: 0.84 (0.74, 0.96) M3: 0.84 (0.75, 0.95)
Exposure: SSSD+SSFD								
1	CTS#  USA	N = 133,477  Excluded: no consent, residents outside California.	Revascularization: including coronary artery bypass grafting and percutaneous	Servings/time Range C1(ref): rare/never	C1(ref): 1,468 C2: 798 C3: 505	Model 1: age  Model 2: model 1 + race/ethnicity.	Model 1; HR (95%CI) C1(ref): 1 C2: 1.01 (0.93, 1.10) C3: 1.03 (0.93, 1.15)	

ROB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Pacheco et al., 2020  20 y  Public funding	incomplete or incomprehensible questionnaires, incomplete dietary intake data, extreme caloric values (<600 or >5000 kcal/d), those aged ≥85 y at baseline, history of CVD and diabetes mellitus.  <b>n</b> = 106,178  <b>Sex:</b> females  <b>Ethnicity:</b> 87.3% Caucasian and 12.7% all other races  <b>Age (mean±SD):</b> 52.1 ± 13.4 y	coronary intervention and/or percutaneous transluminal coronary angioplasty  Annual linkage with state-wide OSHPD hospitalization records, derived medical diagnoses, and in- patient procedures	<b>C2:</b> >rare/never to <1/wk <b>C3:</b> ≥1 /wk to <1 serving/d <b>C4:</b> ≥1 serving/d  <b>Fl. oz/day (mean±SD)</b> <b>C1(ref):</b> 0 ± 0.0 <b>C2:</b> 2.6 ± 0.0 <b>C3:</b> 5.5 ± 0.0 <b>C4:</b> 13.5 ± 0.1  1 fl. oz = 29.6 ml  <b>Serving size</b> = 355 ml  <b>n per categories</b> <b>C1(ref):</b> 43,425 <b>C2:</b> 35,422 <b>C3:</b> 22,825 <b>C4:</b> 4,506  <b>Exposure assessment:</b> SFFQ	<b>C4:</b> 118  <b>Rate per 10,000 person-y</b> <b>C1(ref):</b> 20.0 <b>C2:</b> 12.8 <b>C3:</b> 12.6 <b>C4:</b> 14.9	socioeconomic status, smoking status, alcohol intake, cardiovascular disease family history, physical activity, aspirin use, multivitamin use, menopausal status, menopausal hormone therapy use, oral contraceptive use, and history of hypertension.  <b>Model 3:</b> model 2 + body mass index, total energy intake, and fruit and vegetable intake.  <b>Model 4:</b> age, race/ethnicity, socioeconomic status, smoking status, alcohol intake, cardiovascular disease (CVD) family history, physical activity, aspirin use, menopausal status, menopausal hormone therapy use, history of hypertension, body mass index, and total energy intake	<b>C4:</b> 1.35 (1.12, 1.64) <b>P per trend = 0.006</b>  <b>Model 2; HR (95%CI)</b> <b>C1(ref):</b> 1 <b>C2:</b> 1.03 (0.94, 1.12) <b>C3:</b> 1.03 (0.93, 1.15) <b>C4:</b> 1.24 (1.02, 1.50) <b>P per trend = 0.044</b>  <b>Model 3; HR (95%CI)</b> <b>C1(ref):</b> 1 <b>C2:</b> 1.04 (0.95, 1.14) <b>C3:</b> 1.02 (0.92, 1.14) <b>C4:</b> 1.23 (1.01, 1.50) <b>P per trend = 0.082</b>  <b>Model 4; HR (95%CI)</b> <b>C1(ref):</b> 1 <b>C2:</b> 1.05 (0.96, 1.15) <b>C3:</b> 1.04 (0.94, 1.16) <b>C4:</b> 1.26 (1.04, 1.54) <b>P per trend = 0.037</b>

BMI, body mass index; CABG, coronary artery bypass grafting; CI, confidence interval; CVD, cardiovascular disease; ICD, International Classification of Disease; FFQ, food frequency questionnaire; HR, hazard ratio; HTN, hypertension; MI, myocardial infarction; n, participants analysed; N, participants included in the cohort; PCI, percutaneous coronary intervention; SD, standard deviation; SFFQ, semiquantitative food frequency questionnaire; SSFD, sugar-sweetened fruit drinks; SSSD, sugar-sweetened soft drinks; T2DM, type 2 diabetes mellitus; TS, total sugars; y, year. \*Data provided by authors. † Study identified through an update of the literature search conducted in July 2020. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Incidence of hyperuricemia

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: SSSD</b>							
<b>1</b>	<b>ARIC</b>  USA  Bomback et al. (2010)  3 y  Public funding	<b>N</b> = 15,792  <b>Population sampled:</b> general population from 4 US communities  <b>Excluded:</b> NR  <b>n</b> = 9,451  <b>Sex:</b> 55.2% females  <b>Ethnicity:</b> 72.8% Caucasian, 26.9% Black and 0.3% Other.  <b>Age:</b> 45-64 y	Hyperuricemia was defined using sex-specific cut points of >5.7 mg/dl in women and >7.0 mg/dl in men.  For sensitivity analyses, a gender-neutral definition of hyperuricemia as >7.0 mg/dl was used.	<b>Range (Servings/time)</b> <b>C1 (ref):</b> <1 soda per day <b>C2:</b> 1 soda per day <b>C3:</b> >1 soda per day  1 soda= 240 ml  <b>n per category of intake NR</b>  <b>Exposure assessment:</b> SFFQ	3,288 (34.8%)  <b>n cases per category of intake NR</b>	<b>Model 1:</b> Crude  <b>Model 2:</b> race and centre  <b>Model 3:</b> model 2 + age, sex, caffeine intake, animal protein intake, hypertension, body mass index, renal function, current tobacco and alcohol use	<b>Model 1; OR (95% CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.23 (1.07–1.40) <b>C3:</b> 1.23 (1.02–1.49)  <b>Model 2; OR (95% CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.09 (0.96–1.25) <b>C3:</b> 1.17 (0.97–1.42)  <b>Model 3; OR (95% CI)</b> <b>C1 (ref):</b> 1 <b>C2:</b> 1.11 (0.97–1.28) <b>C3:</b> 1.17 (0.95–1.43)  <b>No relationship was observed for ASSD</b>

ASSD, artificially-sweetened soft drinks; CI, confidence interval; d, day; dl, decilitre; mg, milligrams; n, participants analysed; N, participants included in the cohort; NR, not reported; OR, odds ratio; SFFQ, semiquantitative food frequency questionnaire; SSSD, sugar-sweetened soft drinks; USA, United States of America, y, year. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

## Incidence of gout

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: Fructose</b>							
<b>1</b>	<b>HPFS</b>  USA  Choi and Curhan (2008)  12 y  Mixed funding	<b>N</b> = 51,529  <b>Population sampled:</b> male health professionals  <b>Excluded:</b> history of gout at baseline, incomplete information on intake of SSSD  <b>Follow-up rate:</b> >90% for each two-year period. Those not responding to a questionnaire during one follow-up cycle were not removed from the study  <b>n</b> = 46,393  <b>Sex:</b> males <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 40-75 y	Self-reported in biennial questionnaires plus supplementary questionnaire to ascertain that participants met $\geq 6$ of the 11 survey criteria for gout proposed by the American College of Rheumatology <sup>56</sup> (i.e. >1 attack of acute arthritis, maximum inflammation developed within 1 day, oligoarthritis attack, redness over joints, painful or swollen first metatarsophalangeal joint, unilateral first metatarsophalangeal joint attack, tophus, hyperuricemia, asymmetric swelling within a joint, complete termination of an attack).  Validation against medical records in 50 self-reported cases of gout.  Positive predictive value for incident gout = 94%.	<b>Range (%E)</b> <u>Q1 (ref):</u> <6.9 <u>Q2:</u> 6.9-8.5 <u>Q3:</u> 8.6-10.0 <u>Q4:</u> 10.1-11.8 <u>Q5:</u> >11.8  <b>Person-years</b> <u>Q1 (ref):</u> 87,050 <u>Q2:</u> 87,761 <u>Q3:</u> 87,815 <u>Q4:</u> 88,087 <u>Q5:</u> 87,748  <b>Exposure assessment:</b> SFFQ	<u>Q1 (ref):</u> 186 <u>Q2:</u> 139 <u>Q3:</u> 153 <u>Q4:</u> 137 <u>Q5:</u> 140	<b>Model 1:</b> age, BMI, alcohol and total energy intake  <b>Model 2:</b> model 1 + diuretic use, history of hypertension, history of chronic renal failure, intake of vitamin C and percentage of energy from non-fructose carbohydrate and total protein to estimate effects of substituting fructose for equivalent energy from fat  <b>Model 3:</b> same as model 2 BUT percentage of energy from total carbohydrate to estimate effects of fructose for equivalent energy from other carbohydrates.	<b>Model 1; RR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.90 (0.72-1.13) <u>Q3:</u> 1.11 (0.88-1.39) <u>Q4:</u> 1.08 (0.85-1.37) <u>Q5:</u> 1.24 (0.97-1.57) <b>P per trend 0.04</b>  <b>Model 2; RR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.96 (0.76-1.21) <u>Q3:</u> 1.20 (0.95-1.53) <u>Q4:</u> 1.25 (0.96-1.61) <u>Q5:</u> 1.52 (1.15-2.01) <b>P per trend 0.001</b>  <b>Model 3; RR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.98 (0.77-1.25) <u>Q3:</u> 1.29 (1.00-1.67) <u>Q4:</u> 1.41 (1.06-1.88) <u>Q5:</u> 1.81 (1.31-2.50) <b>P per trend &lt;0.001</b>
<b>2</b>	<b>NHS</b>  USA  Choi et al., 2010  22 y	<b>N</b> = 121,700  <b>Population sampled:</b> female nurses  <b>Excluded:</b> $\geq 10$ food items blank in	Self-reported in biennial questionnaires plus supplementary questionnaire (from 2001) to that participants met $\geq 6$ of the 11 survey criteria for gout proposed by the American College of Rheumatology (see above).	<b>Range (%E)</b> <u>Q1 (ref):</u> <7.5 <u>Q2:</u> 7.51-8.97 <u>Q3:</u> 8.97-10.2 <u>Q4:</u> 10.3-11.9 <u>Q5:</u> >11.9  <b>Person-years</b>	<u>Q1 (ref):</u> 154 <u>Q2:</u> 172 <u>Q3:</u> 149 <u>Q4:</u> 163 <u>Q5:</u> 140	<b>Model 1:</b> age, BMI, alcohol and total energy  <b>Model 2:</b> model 1 + menopause status, hormone therapy, diuretic use, history of hypertension, intake of vitamin C and caffeine, and percentage of energy from non-fructose	<b>Model 1; RR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.01 (0.81-1.27) <u>Q3:</u> 0.87 (0.69-1.10) <u>Q4:</u> 0.98 (0.78-1.24) <u>Q5:</u> 0.98 (0.76-1.25) <b>P per trend 0.80</b>

<sup>56</sup> Wallace SL, Robinson H, Masi AT, Decker JL, McCarty DJ, Yu TF. Preliminary criteria for the classification of the acute arthritis of primary gout. Arthritis Rheum 1977;20:895-900.



	Public funding	the 1984 FFQ, prevalent cases of gout.  <b>Follow-up rate:</b> >90% for each two-year period.  <b>n</b> = 78,906  <b>Sex:</b> females <b>Ethnicity:</b> Caucasian (~93%+) <b>Age:</b> 30 to 55 y	Validation against medical records in 56 self-reported cases of gout.  Positive predictive value for incident gout = 91%.	<u>Q1 (ref):</u> 300,229 <u>Q2:</u> 320,963 <u>Q3:</u> 326,022 <u>Q4:</u> 327,559 <u>Q5:</u> 315,365  <b>Exposure assessment:</b> SFFQ		<i>carbohydrate and total protein to estimate effects of substituting fructose for the equivalent energy from fat.</i>  <b>Model 3:</b> same as model 2 BUT <i>percentage of energy from total carbohydrate to estimate effects of fructose for equivalent energy from other carbohydrates.</i>	<b>Model 2; RR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.14 (0.91-1.44) <u>Q3:</u> 1.02 (0.80-1.31) <u>Q4:</u> 1.18 (0.91-1.53) <u>Q5:</u> 1.18 (0.89-1.56) <b>P per trend 0.27</b>  <b>Model 3; RR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.23 (0.97-1.57) <u>Q3:</u> 1.17 (0.90-1.54) <u>Q4:</u> 1.41 (1.06-1.88) <u>Q5:</u> 1.44 (1.04-2.00) <b>P per trend 0.03</b>
<b>Exposure: free fructose</b>							
<b>1</b>	<b>HPFS</b>  USA  Choi and Curhan (2008)  12 y  Mixed funding	<b>Study population and inclusion criteria as for total fructose</b>	<b>Same ascertainment criteria as for total fructose</b>	<b>Median (range) (%E)</b> <u>Q1 (ref):</u> 2.6 (< 3.5) <u>Q2:</u> 3.8 (3.5-4.4) <u>Q3:</u> 4.7 (4.5-5.3) <u>Q4:</u> 5.8 (5.4-6.6) <u>Q5:</u> 7.9 (> 6.6)  <b>n/ person-years</b> <u>Q1 (ref):</u> 9,278/ 87,136 <u>Q2:</u> 9279/ 87,618 <u>Q3:</u> 9279/ 87,818 <u>Q4:</u> 9279/ 88,050 <u>Q5:</u> 9,278/ 87,839  <b>Exposure assessment:</b> SFFQ	<u>Q1 (ref):</u> 152 <u>Q2:</u> 154 <u>Q3:</u> 146 <u>Q4:</u> 160 <u>Q5:</u> 143	<b>Model 1:</b> age, BMI, alcohol and energy intake  <b>Model 2:</b> model 1 + diuretic use, history of hypertension, history of chronic renal failure, intake of alcohol, vitamin C and <i>percentage of energy from non-fructose carbohydrate and total protein to estimate effects of substituting fructose for equivalent energy from fat</i>  <b>Model 3:</b> same as model 2 BUT <i>percentage of energy from total carbohydrate to estimate effects of fructose for equivalent energy from other carbohydrates.</i>	<b>Model 1; RR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.19 (0.95-1.49) <u>Q3:</u> 1.21 (0.96-1.53) <u>Q4:</u> 1.45 (1.15-1.83) <u>Q5:</u> 1.43 (1.12-1.83) <b>P per trend = 0.001</b>  <b>Model 2; RR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.26 (1.00-1.59) <u>Q3:</u> 1.33 (1.04-1.70) <u>Q4:</u> 1.68 (1.30-2.16) <u>Q5:</u> 1.81 (1.38-2.38) <b>P per trend &lt;0.001</b>  <b>Model 3; RR (95% CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 1.29 (1.02-1.64) <u>Q3:</u> 1.41 (1.09-1.82) <u>Q4:</u> 1.84 (1.40-2.41) <u>Q5:</u> 2.02 (1.49-2.75) <b>P per trend &lt;0.001</b>

2	NHS	Study population and inclusion criteria as for total fructose	Same ascertainment criteria as for total fructose	Range (%E) Q1 (ref): <3.7 Q2: 3.71-4.6 Q3: 4.61-5.45 Q4: 5.46-6.6 Q5: >6.6  n/ person-years Q1 (ref): 21,712/ 294,841 Q2: 15,229/ 320,317 Q3: 13,424/ 327,349 Q4: 12,778/ 329,706 Q5: 15,763/ 317,937  Exposure assessment: SFFQ	Q1 (ref): 132 Q2: 181 Q3: 150 Q4: 160 Q5: 155	Model 1: age, BMI, alcohol and total energy  Model 2: model 1 + menopause status, hormone therapy, diuretic use, history of hypertension, intake of vitamin C and caffeine, and percentage of energy from non-fructose carbohydrate and total protein to estimate effects of substituting fructose for the equivalent energy from fat.  Model 3: same as model 2 BUT percentage of energy from total carbohydrate to estimate effects of fructose for equivalent energy from other carbohydrates.	Model 1; RR (95% CI) Q1 (ref): 1 Q2: 1.13 (0.90-1.42) Q3: 0.91 (0.72-1.16) Q4: 0.99 (0.78-1.26) Q5: 1.14 (0.90-1.45) P per trend 0.52  Model 2; RR (95% CI) Q1 (ref): 1 Q2: 1.25 (0.99-1.58) Q3: 1.07 (0.83-1.37) Q4: 1.21 (0.93-1.56) Q5: 1.43 (1.09-1.88) P per trend 0.02  Model 3; RR (95% CI) Q1 (ref): 1 Q2: 1.31 (1.03-1.66) Q3: 1.15 (0.89-1.50) Q4: 1.34 (1.01-1.76) Q5: 1.62 (1.20-2.19) P per trend 0.004
	USA  Choi et al. (2010)  22 y  Public funding						
Exposure: SSSD							
1	HPFS	Study population and inclusion criteria as for total fructose	Same ascertainment criteria as for total fructose	Range (servings/time) C1 (ref): <1/mo C2: 1/mo-1/wk C3: 2-4/wk C4: 5-6/wk C5: 1/d C6: ≥2/d  Serving size = 12oz (355mL)  n/ person-years C1 (ref): 20,205/ 158,891 C2: 13,247/ 151,173 C3: 4,661/ 53,086 C4: 4, 802/ 47,433 C5: 2,420/ 20,485	C1 (ref): 279 C2: 251 C3: 82 C4: 88 C5: 39 C6: 16	Model 1: age, BMI, alcohol and energy intake  Model 2: model 1 + diuretic use, history of hypertension, history of chronic renal failure, intake of alcohol, total meats, seafood, purine rich vegetables, dairy foods, vitamin C, fruit juice, and diet soft drinks.	Model 1; RR (95% CI) C1 (ref): 1 C2: 1.00 (0.84-1.19) C3: 1.00 (0.78-1.29) C4: 1.30 (1.01-1.67) C5: 1.44 (1.02-2.04) C6: 1.78 (1.06-2.98) P per trend 0.002  Model 2; RR (95% CI) C1 (ref): 1 C2: 1.00 (0.84-1.20) C3: 0.99 (0.77-1.29) C4: 1.29 (1.00-1.68) C5: 1.45 (1.02-2.08) C6: 1.85 (1.08-3.16) P per trend 0.002
	USA  Choi and Curhan (2008)  12 y  Mixed funding						

				C6: 1, 058/ 7,392 <b>Exposure assessment:</b> SFFQ			<b>No relationship was observed for ASD</b>
<b>2</b>	<b>NHS</b> USA Choi et al. (2010) 22 y Public funding	<b>Study population and inclusion criteria as for total fructose</b>	<b>Same ascertainment criteria as for total fructose</b>	<b>Range (servings/time)</b> C1 (ref): <1/mo C2: 1/mo-1/wk C3: 2-4/wk C4: 5-6/wk C5: 1/d C6: ≥2/d  <b>Serving size</b> = 12oz (355mL)  <b>n/ person-years</b> C1 (ref): 41,974/ 789,469 C2: 17,880/ 387,106 C3: 11,766/ 282,172 C4: 2,737/ 66,390 C5: 3,039/ 47,634 C6: 1,510/ 17,379  <b>Exposure assessment:</b> SFFQ	C1 (ref): 383 C2: 187 C3: 129 C4: 35 C5: 31 C6: 13	<b>Model 1:</b> age, BMI, alcohol and total energy intake  <b>Model 2:</b> model 1 + menopausal status, use of hormone therapy, diuretic use, history of hypertension; intake of meat, seafood, dairy products, vitamin C, coffee, fruit juice, and diet soft drinks	<b>Model 1; RR (95% CI)</b> C1 (ref): 1 C2: 1.12 (0.94-1.33) C3: 1.07 (0.88-1.31) C4: 1.42 (1.00-2.02) C5: 2.09 (1.44-3.02) C6: 3.05 (1.74-5.35) <b>P per trend &lt;0.001</b>  <b>Model 2; RR (95% CI)</b> C1 (ref): 1 C2: 1.09 (0.91-1.30) C3: 0.98 (0.79-1.20) C4: 1.25 (0.88-1.79) C5: 1.74 (1.19-2.55) C6: 2.39 (1.34-4.26) <b>P per trend &lt;0.001</b>  <b>No relationship was observed for ASD</b>
<b>Exposure: 100% FJ</b>							
<b>1</b>	<b>HPFS</b> USA Choi and Curhan (2008) 12 y Mixed funding	<b>Study population and inclusion criteria as for total fructose</b>	<b>Same ascertainment criteria as for total fructose</b>	<b>Range (servings/time)<sup>57</sup></b> C1 (ref): <1/mo C2: 1/mo-1/wk C3: 2-4/wk C4: 5-6/wk C5: 1/d C6: ≥2/d  <b>Serving size</b> = 6oz (177mL) <b>Person-years</b> C1 (ref): 26,590 C2: 85,201 C3: 61,964 C4: 107,415	C1 (ref): 31 C2: 137 C3: 116 C4: 191 C5: 236 C6: 43	<b>Model 1:</b> age, BMI, alcohol and energy intake  <b>Model 2:</b> model 1 + diuretic use, history of hypertension, history of chronic renal failure, intake of meat, seafood, purine rich vegetables, dairy foods, vitamin C, sugar-sweetened soft drinks and diet soft drinks	<b>Model 1; RR (95% CI)</b> C1 (ref): 1 C2: 1.37 (0.92-2.02) C3: 1.64 (1.10-2.45) C4: 1.60 (1.09-2.35) C5: 1.76 (1.20-2.57) C6: 1.83 (1.14-2.93) <b>P per trend 0.008</b>  <b>Model 2; RR (95% CI)</b> C1 (ref): 1 C2: 1.34 (0.91-1.99) C3: 1.57 (1.05-2.35) C4: 1.55 (1.05-2.30)

<sup>57</sup> Data refers to total 100% FJ


				<u>C5</u> : 129,859 <u>C6</u> : 26,144  <b>Exposure assessment:</b> SFFQ			<u>C5</u> : 1.74 (1.18-2.56) <u>C6</u> : 1.81 (1.12-2.93) <b>P per trend 0.01</b>
<b>2</b>	<b>NHS</b>  USA  Choi et al. (2010)  22 y  Public funding	<b>Study population and inclusion criteria as for total fructose</b>	<b>Same ascertainment criteria as for total fructose</b>	<b>Range (servings/time)<sup>58</sup></b> <u>C1</u> (ref): <1/mo <u>C2</u> : 1/mo-1/wk <u>C3</u> : 2-4/wk <u>C4</u> : 5-6/wk <u>C5</u> : 1/d <u>C6</u> : ≥2/d  <b>Serving size</b> = 6oz (177mL) <b>Person-years</b> <u>C1</u> (ref): 213,647 <u>C2</u> : 346,219 <u>C3</u> : 506,760 <u>C4</u> : 268,532 <u>C5</u> : 236,894 <u>C6</u> : 18,099  <b>Exposure assessment:</b> SFFQ	<u>C1</u> (ref): 71 <u>C2</u> : 145 <u>C3</u> : 277 <u>C4</u> : 171 <u>C5</u> : 103 <u>C6</u> : 11	<b>Model 1:</b> age, BMI, alcohol and total energy intake  <b>Model 2:</b> model 1 + menopausal status; use of hormone therapy; diuretic use; history of hypertension; intake of total meats, seafood, dairy products, vitamin C, coffee, sugar-sweetened soft drinks and diet soft drinks	<b>Model 1; RR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 1.33 (1.00-1.77) <u>C3</u> : 1.39 (1.07-1.81) <u>C4</u> : 1.59 (1.20-2.10) <u>C5</u> : 1.48 (1.09-2.01) <u>C6</u> : 2.52 (1.33-4.77) <b>P per trend 0.008</b>  <b>Model 2; RR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 1.27 (0.95-1.69) <u>C3</u> : 1.30 (0.99-1.70) <u>C4</u> : 1.50 (1.12-2.00) <u>C5</u> : 1.41 (1.03-1.93) <u>C6</u> : 2.42 (1.27-4.63) <b>P per trend 0.02</b>

ASSD, artificially-sweetened soft drinks; BMI, body mass index; CI, confidence interval; d, day; E, energy; FFQ, food frequency questionnaire; FJ, fruit juice; ml, millilitres; mo, month; n, participants analysed; N, participants included in the cohort; oz, ounces; RR, risk ratio; SFFQ, semiquantitative food frequency questionnaire; SSSD, sugar-sweetened soft drinks; USA, United States of America, wk, week; y, year. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

<sup>58</sup> Data refers to orange juice, which in this population is the major contributor among juices to free fructose intake (17%). Data for total 100% FJ not reported.

## Observational studies on pregnancy endpoints

### Incidence of gestational diabetes mellitus (GDM)

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: total sugars</b>							
<b>3</b>	<b>ALSWH<sup>59</sup></b>  Australia  Looman et al. (2018)  12 y  Public funding	<b>N</b> = 40,000 approx.  <b>Pop. sampled:</b> Women from Australia's national health care system  <b>Excluded:</b> no report of a live birth in 2006, 2009, 2012, 2015; missing data on diet on 2003 and 2009; missing data on GDM; ratio of reported energy intake and predicted energy requirement <0.56 or >1.44; history of T1DM or T2DM before GDM diagnosis; history of GDM before baseline (2003); missing covariate data.  <b>n</b> = 3,607 (6,263 pregnancies) <b>Ethnicity:</b> Caucasian <b>Age:</b> 25-30 y	Self-reported physician diagnosis of GMD. Diagnosis was confirmed after a 75-g OGTT with plasma glucose at 0 h ≥5.5 mmol/l and/or at 2 h ≥8.0 mmol/l <sup>60</sup> . Diagnostic criteria were updated in 2013 (plasma glucose at 0 h ≥5.1 mmol/l and/or ≥10.0 mmol/l at 1 h and/or ≥8.5 mmol/l at 2h <sup>61</sup> ).  PPV of self-reported incident GDM = 91% as compared to medical records in a validation study including 1,914 women <sup>62</sup> .	<b>g/d (median)</b>  <u>Q1 (ref):</u> 59.6 <u>Q2:</u> 76.1 <u>Q3:</u> 89.0 <u>Q4:</u> 106.2  <b>n women/pregnancies</b> <u>Q1 (ref):</u> 901/1,541 <u>Q2:</u> 903/1,606 <u>Q3:</u> 902/1,586 <u>Q4:</u> 901/1,530  <b>Exposure assessment:</b> SFFQ	<b>GDM cases (pregnancies)/% of total pregnancies</b>  <u>Q1 (ref):</u> 90/ 5.8 <u>Q2:</u> 71/ 4.4 <u>Q3:</u> 61/ 3.9 <u>Q4:</u> 63/ 4.1	<b>Model 1:</b> age at pregnancy, country of birth, educational level, total energy intake, physical activity, smoking, polycystic ovarian syndrome, hypertension during pregnancy, parity, inter-pregnancy interval.  <b>Model 2:</b> model 1 + fat and protein intake (E%).  <b>Model 3:</b> model 2 + BMI.	<b>Model 1; RR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.78 (0.58, 1.06) <u>Q3:</u> 0.71 (0.51, 0.99) <u>Q4:</u> 0.72 (0.52, 0.99) <b>P for trend = 0.04</b>  <b>Model 2; RR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.83 (0.61, 1.13) <u>Q3:</u> 0.78 (0.54, 1.13) <u>Q4:</u> 0.83 (0.56, 1.24) <b>P for trend = 0.33</b>  <b>Model 3; RR (95%CI)</b> <u>Q1 (ref):</u> 1 <u>Q2:</u> 0.83 (0.61, 1.14) <u>Q3:</u> 0.77 (0.54, 1.11) <u>Q4:</u> 0.83 (0.56, 1.23) <b>P for trend = 0.32</b>
<b>Exposure: SSSD</b>							

<sup>59</sup> The ALSWH also reports on the exposure sugars added to foods and beverages by the consumer; data not extracted.


<sup>60</sup> Hoffman L, Nolan C, Wilson JD, et al. (1998) Gestational diabetes mellitus – management guidelines. The Australasian Diabetes in Pregnancy Society. Med J Aust 169, 93–97.

<sup>61</sup> Nankervis A, McIntyre H, Moses R, et al. (2013) ADIPS consensus guidelines for the testing and diagnosis of gestational diabetes mellitus in Australia. <http://adips.org/downloads/ADIPSConsensusGuidelinesGDM-03.05.13VersionACCEPTED FINAL.pdf>

<sup>62</sup> Gresham E, Forder P, Chojenta CL, et al. (2015) Agreement between self-reported perinatal outcomes and administrative data in New South Wales, Australia. BMC Pregnancy Childbirth 15, 161.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
1	<b>SUN</b>  Spain  Donazar-Ezcurra et al. (2018)  10.3 y (mean)  Public funding	<b>N</b> = 13,777  <b>Excluded:</b> no report of a live birth during follow-up, reporting exceedingly low or high (<1th or >99th percentiles) total energy intake, diagnoses of diabetes or previous GDM  <b>Follow-up rate:</b> 91%  <b>n</b> = 3,396 <b>Ethnicity:</b> Caucasian <b>Age</b> (mean $\pm$ SD) <u>C1</u> (ref): 29.5 $\pm$ 5.3 y <u>C2</u> : 28.5 $\pm$ 4.7 y <u>C3</u> : 27.9 $\pm$ 4.2 y <u>C4</u> : 28.1 $\pm$ 4.4 y	Self-reported incidence of GDM in biennial questionnaires. Reported cases were verified by a committee of medical doctors based on additional information requested to the participant through a questionnaire.  <u>Diagnostic criteria for GDM:</u> 2-step approach (a 50-g OGTT plus a 100-g OGTT if plasma glucose > 7.8 mmol/l) at 24-28 weeks of gestation, using the cut-offs of the American Diabetes Association for a positive 100-g OGTT <sup>63</sup> .  Positive predicted value of self-reported GDM = 80%	<b>Servings/time (range)</b>  <u>C1</u> (ref): $\leq 1$ /mo (rarely or never) <u>C2</u> : 1-3/mo <u>C3</u> : >1-3/mo- $\leq 1$ /wk <u>C4</u> : $\geq 2$ /wk  Serving size = 200 ml  <b>n</b> <u>C1</u> (ref): 831 <u>C2</u> : 808 <u>C3</u> : 795 <u>C4</u> : 962  <b>Exposure assessment:</b> SFFQ	<u>C1</u> (ref): 29 <u>C2</u> : 41 <u>C3</u> : 41 <u>C4</u> : 61	<b>Model 1:</b> age.  <b>Model 2:</b> model 1 + BMI, family history of diabetes, current smoking status, physical activity, parity, fast-food consumption, Mediterranean dietary score, alcohol intake, multiple pregnancy, CVD/hypertension at baseline, fibre intake, following special diet and snacking, total energy intake.  <b>Model 3:</b> model 2 without adjustment for total energy intake.	<b>Model 1; OR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 1.56 (0.96, 2.54) <u>C3</u> : 1.64 (1.00, 2.68) <u>C4</u> : 2.02 (1.28, 3.19) <b>P for trend = 0.003</b>  <b>Model 2; OR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 1.55 (0.94, 2.55) <u>C3</u> : 1.67 (1.01, 2.77) <u>C4</u> : 2.03 (1.25, 3.31) <b>P for trend = 0.006</b>  <b>Model 3; OR (95% CI)</b> <u>C1</u> (ref): 1 <u>C2</u> : 1.56 (0.95, 2.56) <u>C3</u> : 1.68 (1.02, 2.78) <u>C4</u> : 2.06 (1.28, 3.34) <b>P for trend = 0.004</b>
<b>Exposure: SSSD+SSFD</b>							
2	<b>NHS II</b>  USA	<b>N</b> = 116,671  <b>Excluded:</b> SFFQ not completed in 1991, >70	Self-reported incidence of GDM in biennial questionnaires.	<b>Servings/time (range)</b>  <u>C1</u> (ref): 0-3/mo <u>C2</u> : 1-4/wk	<u>C1</u> (ref): 423 <u>C2</u> : 229 <u>C3</u> : 208	<b>Model 1:</b> age and parity  <b>Model 2:</b> model 1 + race/ethnicity, cigarette smoking status, family	<b>Model 1, RR (95%CI)</b> <u>C1</u> (ref): 1.00 <u>C2</u> : 1.01 (0.85,1.20) <u>C3</u> : 1.23 (1.05,1.45)

<sup>63</sup> American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. Diabetes Care 2010;33 Suppl1:S62-9. doi: 10.2337/dc10-S062

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Chen et al. (2009)  10 y  Public funding	items in the FFQ were left blank, reporting of implausible total energy intake (<500 kcal/day or > 3500 kcal/day), reporting multiple gestation, no physical activity data in 1991, history of diabetes, cancer, CVD or GDM reported in 1989 or 1991.  <b>Follow-up rate:</b> 90% (approx.) for every 2-y period  <b>n</b> = 13,475  <b>Ethnicity:</b> Caucasian (~90%+)  <b>Age:</b> 22-44 y	Medical records reviewed among a sample of 114 women in the cohort who corroborated on a supplementary questionnaire that they had at first diagnosis of GDM in a singleton pregnancy between 1989 and 1991. Of these 94% had a physician diagnosis.	<u>C3</u> : ≥5/wk  Serving size = 12oz (334 mL) <sup>64</sup>  <b>n/person-years</b> <u>C1</u> (ref): 5,584/185,682 <u>C2</u> : 3,675/173,189 <u>C3</u> : 4,216/185,757  <b>Exposure assessment:</b> SFFQ		history of diabetes in a first-degree relative, alcohol intake and physical activity  <b>Model 3:</b> model 2 + BMI  <b>Model 4:</b> model 3 + Western dietary pattern score	<b>P for trend = 0.005</b>  <b>Model 2, RR (95%CI)</b> <u>C1</u> (ref): 1.00 <u>C2</u> : 1.02 (0.86,1.21) <u>C3</u> : 1.17 (1.00,1.37) <b>P for trend = 0.04</b>  <b>Model 3, RR (95%CI)</b> <u>C1</u> (ref): 1.00 <u>C2</u> : 1.06 (0.89,1.25) <u>C3</u> : 1.23 (1.05,1.44) <b>P for trend = 0.01</b>  <b>Model 4, RR (95%CI)</b> <u>C1</u> (ref): 1.00 <u>C2</u> : 1.03 (0.87,1.23) <u>C3</u> : 1.16 (0.98,1.37) <b>P for trend = 0.06</b>  <b>RR (95% CI) per each serving increase per day</b> <u>Model 1</u> : 1.25 (1.07,1.45) <u>Model 2</u> : 1.18 (1.01,1.37) <u>Model 3</u> : 1.23 (1.05,1.43) <u>Model 4</u> : 1.16 (0.99,1.36)
<b>Exposure: TFJ</b>							
<b>3</b>	<b>ALSWH</b>  Australia  Looman et al. (2018)  12 y	<b>Same population and exclusion criteria as for total sugars</b>	<b>Same ascertainment of outcome as for total sugars</b>	<b>g/d</b>   NR  <b>Exposure assessment:</b> SFFQ	<b>NR</b>	<b>Model 1:</b> age, country of birth, educational level, total energy intake, physical activity, smoking, polycystic ovarian syndrome, hypertension during pregnancy, parity, inter-pregnancy interval.  <b>Model 2:</b> model 1 + intake of other carbohydrate food groups (i.e. white bread, high-fibre bread, cereal, fruit,	<b>Per each 100 g/d increase</b> <b>RR (95%CI)</b> <u>Model 1</u> : 0.88 (0.79, 0.99) <u>Model 2</u> : 0.89 (0.79, 0.99) <u>Model 3</u> : 0.89 (0.80, 1.00) <b>P for trend = 0.01</b>

<sup>64</sup> Cohen L, Curhan G, Forman J (2012) Association of sweetened beverage intake with incident diabetes. J Gen Intern Med 27(9):1127–34

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
	Public funding					fruit juice, staple products, vegetables).  <b>Model 3:</b> model 2 + BMI.	
<b>Exposure: 100% FJ</b>							
<b>2</b>	<b>NHS II</b>  USA  Chen et al. (2012)  10 y  Public funding	<b>N</b> = 116,671  <b>Excluded:</b> SFFQ not completed in 1991, >70 items in the FFQ were left blank, reporting of implausible total energy intake (<500 kcal/day or > 3500 kcal/day), reporting multiple gestation, no physical activity data in 1991, history of diabetes, cancer, CVD or GDM reported in 1989 or 1991.  <b>n</b> = 13,475 <b>Ethnicity:</b> Caucasian (~90%+) <b>Age:</b> 24-44 y	Self-reported incidence of GDM in biennial questionnaires. Medical records reviewed among a sample of 114 women in the cohort who corroborated on a supplementary questionnaire that they had at first diagnosis of GDM in a singleton pregnancy between 1989 and 1991. Of these 94% had a physician diagnosis.	<b>servings/d (median)</b> <b>Q1:</b> 0.10 <b>Q2:</b> 0.28 <b>Q3:</b> 0.57 <b>Q4:</b> 1 <b>Q5:</b> 1.72  Serving size = 6oz (167mL)  <b>Person-years</b> <b>Q1:</b> 119,393 <b>Q2:</b> 114,957 <b>Q3:</b> 98,842 <b>Q4:</b> 103,228 <b>Q5:</b> 108,209  <b>Exposure assessment:</b> SFFQ	<b>Q1:</b> 248 <b>Q2:</b> 146 <b>Q3:</b> 148 <b>Q4:</b> 154 <b>Q5:</b> 164	<b>Model 1:</b> age and parity  <b>Model 2:</b> model 1 + race/ethnicity, cigarette smoking status, family history of diabetes in a first-degree relative, alcohol intake and physical activity, BMI  <b>Model 3:</b> model 2 + intake of cereal fiber, processed meat, red meat, SSBs	<b>Model 1, RR (95%CI)</b> <b>Q1 (ref):</b> 1.00 <b>Q2:</b> 0.82 (0.67,1.01) <b>Q3:</b> 0.73 (0.59,0.89) <b>Q4:</b> 0.74 (0.60,0.90) <b>Q5:</b> 0.83 (0.68,1.01) <b>P for trend = 0.06</b>  <b>Model 2, RR (95%CI)</b> <b>Q1 (ref):</b> 1.00 <b>Q2:</b> 0.85 (0.69,1.05) <b>Q3:</b> 0.79 (0.64,0.97) <b>Q4:</b> 0.85 (0.69,1.04) <b>Q5:</b> 1.00 (0.81,1.22) <b>P for trend = 0.93</b>  <b>Model 3, RR (95%CI)</b> <b>Q1:</b> 1.00 (Ref) <b>Q2:</b> 0.82 (0.66, 1.01) <b>Q3:</b> 0.78 (0.63, 0.96) <b>Q4:</b> 0.84 (0.68, 1.04) <b>Q5:</b> 1.00 (0.81, 1.23) <b>P for trend = 0.76</b>

BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; d, day; FFQ, food frequency questionnaire; FJ, fruit juice; FPG, fasting plasma glucose; GDM, gestational diabetes mellitus; h, hour; mo, month; n, participants analysed; N, participants included in the cohort; OGTT, oral glucose tolerance test; OR, odds ratio; RR, risk ratio; SFFQ, semiquantitative food frequency questionnaire; SSB, sugar-sweetened beverages; SSFD, sugar-sweetened fruit drinks; SSSD, sugar-sweetened soft drinks; T1DM, type 1 diabetes mellitus; T2DM, type 2 diabetes mellitus; USA, United States of America; wk, week; y, year. † Exposure adjusted for total energy intake using the residual method (Willett, 1997) *Unless otherwise noted, all of the above cohorts are prospective cohorts.*



## Birthweight related outcomes

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
<b>Exposure: Total Sugars</b>							
<b>1</b>	<b>HSS-USA</b>  USA  Crume et al. (2016)  Public funding	<b>N</b> = 1,410  <b>Excluded:</b> women who had been diagnosed with GDM and neonates born at less than 32 weeks gestation or those without body composition measures at birth  <b>n</b> = 1,040 <b>Ethnicity:</b> White 54.81%, Hispanic 24.62%, Black 14.71%, Other 5.87%  <b>Age:</b> >16 y, mean $\pm$ SD: 27.87 $\pm$ 6.11 y	<b>Birth weight</b> (continuous) measured by trained nurses	<b>g/d [median (IQR)]</b> 107.72 (85, 135.57)  <b>Exposure assessment:</b> one 24-h dietary recall every month during pregnancy (82% had 2 or more)		<b>Model 1:</b> infant sex, gestational age at birth, postnatal age at outcome measurement, maternal age, gravidity, race/ethnicity, smoking at any time during pregnancy and physical activity levels during pregnancy + TEI (energy substitution model) or energy from other macronutrients (energy partition model)  <b>Model 2:</b> model 1 + pre-pregnancy BMI	<b><u>Energy substitution model</u></b>  <b>Per each 1%E increase <math>\beta</math> coefficient (95% CI), g</b>  <b>Model 1</b> -3.24 (-8.73, 2.25), p = 0.2  <b>Model 2</b> -2.32 (-7.78, 3.14), p = 0.4  <b><u>Energy partition model</u></b>  <b>Per 100kcal/day increase <math>\beta</math> coefficient (95% CI), g</b>  <b>Model 1</b> -4.51 (-19.40, 10.37), p = 0.6  <b>Model 2</b> -4.51 (-19.40, 10.38), p = 0.5

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
2	Camden  USA  Lenders et al. (1997)  Followed during pregnancy  Public funding	<b>N</b> = NR  <b>Pop. sampled:</b> Pregnant adolescents  <b>Excluded:</b> women with chronic or metabolic diseases that could affect maternal growth, nutritional status or fetal outcome; intravenous drug use or cocaine addiction; heavy drinkers (>50 g/d) or smokers (>2packs/d), carrying multiple pregnancies, history of diabetes or GDM in current pregnancy  <b>n</b> = 594 <b>Ethnicity:</b> 61% Black, 30% Hispanic and 9% White <b>Age:</b> 12-19 y	<b>SGA</b> = <10th percentile of birth weight for gestational age <sup>65</sup>  <b>LBW</b> = birth weight <2,500 g	<b>g/d</b> <b>G1 (ref):</b> <206 g/d Unadjusted intake (mean ±SD): 111 ±46 Energy adjusted intake (least square means ±SEM): 115 ±2  <b>G2:</b> ≥ 206 g/d Unadjusted intake (mean ±SD): 267 ±73 Energy adjusted intake ((least square means ± SEM)): 227 ± 6  206 g/d = cut off for the 90 <sup>th</sup> percentile of total sugars intake  <b>n</b> <b>G1 (ref):</b> 534 <b>G2:</b> 60  <b>Exposure assessment:</b> 24-h dietary recall at entry and at 28 and 36 weeks of gestation	<b>SGA (n (%))</b> <b>G1 (ref):</b> 37 (7%) <b>G2:</b> 8 (13%)  <b>LBW (n (%))</b> <b>G1:</b> 49 (9%) <b>G2:</b> 10 (17%)	<b>Model:</b> ethnicity, age, number of cigarettes smoked/d, inadequate weight gain, BMI, total energy intake, low gynaecological age, parity, pregnancy-induced hypertension, and inadequate prenatal care.	<b>SGA OR (95% CI)</b> 2.01 (1.05,7.53)  <b>LBW</b> no logistic regression analysis available
<b>Exposure: SSSD</b>							
1	MoBa  Norway  Grundt et al. (2017)  Public funding	<b>N</b> = 75,075 mother-child dyads  <b>Excluded:</b> premature or post-term births, significant malformations, energy intakes considered probably erroneous (< 4.5 MJ or > 20 MJ/day), eating disorders in pregnancy, pre-existing diabetes, missing data on covariates	<b>Birthweight</b> was measured immediately after birth by midwives.  <b>LBW</b> = birth weight <2,500 g	<b>ml/day (range)</b>  <b>C1:</b> <100 <b>C2:</b> 100-500 <b>C3:</b> ≥500  <b>n (no GDM)</b> <b>C1:</b> 38,459 <b>C2:</b> 12,986	<b>LBW</b>  No GDM: 356 GDM: 1  <b>HBW</b>  No GDM: 1,793	<b>Model 1:</b> crude  <b>Model 2:</b> maternal height, pre-pregnancy BMI, age, parity, education and income, diet pattern, exercise, smoking, volume of alcohol intake per occasion prior to pregnancy,	<b>Linear regression analysis Birthweight</b>  <b>Per 100 ml/day increase β coefficient (95% CI), g</b>  <b>no GDM</b> <b>Model 1:</b> -6.0 (-8.2, -3.9) <b>Model 2:</b> -7.8 (-10.3, -5.3)

<sup>65</sup> Brenner, W. E., Edelman, D. A. & Hendricks, C. H. (1976). A standard for foetal growth for the United States of America. Am. J. Obstet. Gynecol. 126: 555–564.

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
		<p><b>n</b></p> <p><b>No GDM:</b> 50,280 <b>GDM:</b> 432</p> <p><b>Ethnicity:</b> Caucasian</p> <p><b>Age (mean <math>\pm</math> SD):</b>  <u>C1</u>: 30.7 <math>\pm</math> 4.3 y  <u>C2</u>: 28.9 <math>\pm</math> 4.5 y  <u>C3</u>: 27.9 <math>\pm</math> 5.0 y</p>	<p><b>HBW</b> = birth weight &gt;4,500 g</p> <p><b>SGA</b> = &lt;10th percentile of birth weight for gestational age according to Norwegian percentiles<sup>66</sup>.</p> <p><b>LGA</b> = &gt;90th percentile of birth weight for gestational age according to Norwegian percentiles.</p>	<p><u>C3</u>: 1,706</p> <p><b>n (GDM)</b>  <u>C1</u>: 454  <u>C2</u>: 81  <u>C3</u>: 15</p> <p><b>Exposure assessment:</b>  SFFQ at week 22 of pregnancy for whole diet; questionnaires at weeks 15, 22 and 30 of pregnancy for beverages. Data analysed for mean intakes</p>	GDM: 36	<p>ASSD intake, spontaneous labour, offspring year of birth.</p> <p><i>Multiple regression models were built using manual forward stepwise procedure. Confounders were considered for inclusion if they were associated with both SSSD and birth weight with a p-value &lt; 0.1.</i></p>	<p><b>GDM</b>  <b>Model 1:</b> 15.4 (-9.5, 40.3)  <b>Model 2:</b> 25.1 (-2.0, 52.2)</p> <p><i>ASSD were significantly negatively associated with birth weight in women with no GDM. The magnitude of their estimated association was 50% of that of SSSD.</i></p> <p><b><u>Multinomial logistic regression analysis</u></b>  <b>Per 100 ml/day increase</b></p> <p><b>LBW, OR (95%CI)</b></p> <p><b>no GDM</b>  <b>Model 1:</b> 1.08 (1.04, 1.12)  <b>Model 2:</b> 1.05 (0.99, 1.10)</p> <p><b>HBW, OR (95%CI)</b></p> <p><b>no GDM</b>  <b>Model 1:</b> 0.98 (0.95, 1.01)  <b>Model 2:</b> 0.94 (0.90, 0.97)</p> <p><b>GDM</b>  <b>Model 1:</b> 1.10 (0.96, 1.25)  <b>Model 2:</b> 1.18 (1.00, 1.39)</p> <p><i>Results reported to be similar for SGA and LGA, respectively, but not shown in the paper</i></p>

<sup>66</sup> Skjaerven, R., Gjessing, H. K., & Bakketeig, L. S. (2000). Birthweight by gestational age in Norway. Acta Obstetrica et Gynecologica Scandinavica, 79, 440–449

RoB Tier	Cohort name Country Reference Follow-up Funding	Original cohort (N total) Exclusion criteria Study population (n, sex and age at baseline)	Ascertainment of outcome	Exposure groups n/person-years Exposure assessment method	Incident cases	Model covariates	Results
2	<b>GeliS<sup>67</sup></b>  Germany  Günther et al. (2019)  Public funding	<b>N</b> = 2,286  <b>Excluded:</b> incomplete data on relevant infant parameters, invalid questionnaires, under- and over-reporters, multiple or complicated pregnancies, diagnosis of severe illnesses  <b>n</b>  <b>Early pregnancy (≤12<sup>th</sup> wk of gestation):</b> 1,902 <b>Late pregnancy (&gt;29<sup>th</sup> wk of gestation):</b> 1,861 <b>Ethnicity:</b> Caucasian <b>Age:</b> 18 – 43 y	<b>Birthweight</b> was retrieved from birth records collected from medical practices.  LBW = birth weight <2,500 g  HBW = birth weight >4,000 g  SGA = <10th percentile of birth weight for gestational age  LGA = >90th percentile of birth weight for gestational age	<b>ml/day = NR</b>  <b>Exposure assessment:</b> SFFQ at or before week 12 wk of gestation and again after week 29.	<b>NR</b>	<b>Model:</b> pre-pregnancy BMI, age, parity and group assignment.	<b>Linear regression analysis Birthweight</b>  <b>Per 200 ml/day increase β coefficient (95% CI), g</b>  <b>Early pregnancy:</b> -10.90 (-18.17, -3.64)  <b>Late pregnancy:</b> -8.19 (-16.26, -0.11)  <b>Binary logistic regression Per 200 ml/day increase</b>  <b>LBW, OR (95% CI)</b> <b>Early pregnancy:</b> 1.04 (0.99,1.09) <b>Late pregnancy:</b> 1.01 (0.94,1.09)  <b>HBW, OR (95% CI)</b> <b>Early pregnancy:</b> 0.95 (0.88,1.02) <b>Late pregnancy:</b> 0.95(0.88,1.03)  <b>SGA, OR (95% CI)</b> <b>Early pregnancy:</b> 1.03(0.99,1.08) <b>Late pregnancy:</b> 1.00(0.94,1.07)  <b>LGA, OR (95% CI)</b> <b>Early pregnancy:</b> 0.94 (0.87,1.02) <b>Late pregnancy:</b> 0.95 (0.87,1.03)

ASSD, artificially sweetened soft drink; BMI, body mass index; BW, body weight; CI, confidence interval; D, day; GDM, gestational diabetes mellitus; h, hour; HBW, high birth weight; IQR, interquartile range; LBW, low birth weight; LGA, large for gestational age; n, participants analysed; N, participants included in the cohort; NR, not reported; OR, odds ratio; SD, standard deviation; SE, standard error; SFFQ, semiquantitative food frequency questionnaire; SGA, small for gestational age; SSC, sugar-sweetened carbonated soft drinks; SSSD, sugar-sweetened soft drinks; TEI, total energy intake; USA, United States of America; y, year. *Unless otherwise noted, all of the above cohorts are prospective cohorts.*

<sup>67</sup> This study also reports on another relevant exposure, sucrose, but only results on SSBs are extracted, which is in line with the approach for considering studies from the update of the literature search.

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